

Michael Potegal
Gerhard Stemmler
Charles Spielberger
Editors

International Handbook of Anger

Constituent and Concomitant Biological,
Psychological, and Social Processes



Springer

International Handbook of Anger

Michael Potegal · Gerhard Stemmler · Charles Spielberger
Editors

International Handbook of Anger

Constituent and Concomitant Biological,
Psychological, and Social Processes



Springer

Editors

Michael Potegal
Department of Pediatrics
University of Minnesota
Division of Pediatric Clinical
Neuroscience
420 Delaware St. SE.
Minneapolis MN 55455
USA
poteg001@umn.edu

Gerhard Stemmler
Universität Marburg
FB Psychologie
Gutenbergstr. 18
35032 Marburg
Germany
gerhardstemmler@online.de

Charles Spielberger
University of South Florida
140 7th Ave. South
Cpy Petersburg FL 33701
USA
spielber@cas.usf.edu

ISBN 978-0-387-89675-5 e-ISBN 978-0-387-89676-2

DOI 10.1007/978-0-387-89676-2

Springer New York Dordrecht Heidelberg London

Library of Congress Control Number: 2009942529

© Springer Science+Business Media, LLC 2010

All rights reserved. This work may not be translated or copied in whole or in part without the written permission of the publisher (Springer Science+Business Media, LLC, 233 Spring Street, New York, NY 10013, USA), except for brief excerpts in connection with reviews or scholarly analysis. Use in connection with any form of information storage and retrieval, electronic adaptation, computer software, or by similar or dissimilar methodology now known or hereafter developed is forbidden.

The use in this publication of trade names, trademarks, service marks, and similar terms, even if they are not identified as such, is not to be taken as an expression of opinion as to whether or not they are subject to proprietary rights.

Printed on acid-free paper

Springer is part of Springer Science+Business Media (www.springer.com)

Contents

Part I Introduction

- 1 **Cross-Disciplinary Views of Anger: Consensus and Controversy** 3
Michael Potegal and Gerhard Stemmler
- 2 **A Brief History of Anger** 9
Michael Potegal and Raymond W. Novaco

Part II Biology of Anger

- 3 **Population and Molecular Genetics of Anger and Aggression: Current State of the Art** 27
Martin Reuter
- 4 **Constructing a Neurology of Anger** 39
Michael Potegal and Gerhard Stemmler
- 5 **Anger, Motivation, and Asymmetrical Frontal Cortical Activations** 61
Eddie Harmon-Jones, Carly K. Peterson, and Cindy Harmon-Jones
- 6 **The Neurochemistry and Psychopharmacology of Anger** 79
Alyson J. Bond and Janet Wingrove
- 7 **Somatovisceral Activation During Anger** 103
Gerhard Stemmler

Part III Cross Cultural Expressions and Experience of Anger

- 8 **The Expression of Anger Across Cultures** 125
David Matsumoto, Seung Hee Yoo, and Joanne Chung
- 9 **Vocal Expressions of Anger** 139
James A. Green, Pamela G. Whitney, and Gwen E. Gustafson
- 10 **Cross-Cultural Experience of Anger: A Psycholinguistic Analysis** 157
Zoltán Kövecses

Part IV Anger in Child Development

- 11 **The Development of Anger** 177
Michael Lewis

12	Anger in Children's Tantrums: A New, Quantitative, Behaviorally Based Model	193
	Michael Potegal and Peihua Qiu	
13	The Development of Anger from Preschool to Middle Childhood: Expressing, Understanding, and Regulating Anger	219
	Elizabeth A. Lemerise and Bridgette D. Harper	
14	Anger and the Reactive-Proactive Aggression Distinction in Childhood and Adolescence	231
	Julie A. Hubbard, Lydia J. Romano, Meghan D. McAuliffe, and Michael T. Morrow	
Part V Appraisal and Information Processing in Anger		
15	Why Do I Get Angry? A Componential Appraisal Approach	243
	Tanja Wranik and Klaus R. Scherer	
16	Appraisals and Anger: How Complete Are the Usual Appraisal Accounts of Anger?	267
	Leonard Berkowitz	
17	Fuel in the Fire: How Anger Impacts Judgment and Decision-Making	287
	Paul M. Litvak, Jennifer S. Lerner, Larissa Z. Tiedens, and Katherine Shonk	
18	State and Trait Anger, Fear, and Social Information Processing	311
	David Schultz, Angela Grodack, and Carroll E. Izard	
Part VI Lifespan Changes and Gender Differences in Anger		
19	The Sociological Study of Anger: Basic Social Patterns and Contexts	329
	Scott Schieman	
20	Anger in the Context of Gender	349
	Agneta H. Fischer and Catharine Evers	
21	Madmen: An Evolutionary Perspective on Anger and Men's Violent Responses to Transgression	361
	Daniel M.T. Fessler	
Part VII Time Course of Anger: State, Trait and the Persistence of Vengefulness		
22	The Temporal Dynamics of Anger: Phenomena, Processes, and Perplexities	385
	Michael Potegal	
23	The Nature and Measurement of Anger	403
	Charles D. Spielberger and Eric C. Reheiser	
24	"Righteous" Anger and Revenge in the Workplace: The Fantasies, the Feuds, the Forgiveness	413
	Thomas M. Tripp and Robert J. Bies	
Part VIII Clinical Aspects of Anger		
25	Anger/Hostility and Cardiovascular Disease	435
	Janice E. Williams	

26	The Anger of Pain Sufferers: Attributions to Agents and Appraisals of Wrongdoings	449
	Ephrem Fernandez and Ajay Wasan	
27	Anger and Psychopathology	465
	Raymond W. Novaco	
28	Toward an Integrative Psychotherapy for Maladaptive Anger	499
	Ephrem Fernandez	
Part IX Conflict and Anger in Family, Workplace, and Community		
29	The Contribution of Child Anger and Fear, and Parental Discipline to Early Antisocial Behavior: An Integrative Model	517
	James Snyder, Lynn Schrepferman, Amber McEachern, and Jamie DeLeeuw	
30	Anger in Intimate Relationships	535
	Donald G. Dutton	
31	Don't Worry, Be Angry? Effects of Anger on Feelings, Thoughts, and Actions in Conflict and Negotiation	545
	Gerben A. Van Kleef	
32	Anger, Violence, and Political Science	561
	Roger Petersen and Sarah Zukerman	
	Subject Index	583

Contributors

Leonard Berkowitz University of Wisconsin-Madison, Madison, WI 53705, USA,
lberkowi@wisc.edu

Robert J. Bies Georgetown University, Georgetown, WA, USA, biesr@georgetown.edu

Alyson J. Bond Institute of Psychiatry, King's College London, London, UK,
a.bond@iop.kcl.ac.uk

Joanne Chung San Francisco State University, San Francisco, CA, USA,
joanne.chung@davidmatsumoto.info

Jamie DeLeeuw Wichita State University, Wichita, KS, USA, jldeleeuw@wichita.edu

Donald G. Dutton Department of Psychology, University of British Columbia, Vancouver, BC,
Canada, dondutton@shaw.ca

Catharine Evers Utrecht University, Utrecht, The Netherlands, c.evers@uu.nl

Ephrem Fernandez Department of Psychology, University of Texas at San Antonio, San Antonio,
TX 78249, USA, ephrem.fernandez@utsa.edu

Daniel M.T. Fessler Department of Anthropology, Center for Behavior, Evolution, and Culture,
University of California, Los Angeles, CA 90095-1553, USA, dfessler@anthro.ucla.edu

Agneta Fischer Department of Social Psychology, University of Amsterdam, 1018 WB,
Amsterdam, The Netherlands, a.h.fischer@uva.nl

James A. Green Department of Psychology, U-1020, University of Connecticut, Storrs, CT
06268, USA, james.green@uconn.edu

Angela Grodack University of Maryland – Baltimore County, Baltimore, MD 21250, USA,
grodack1@umbc.edu

Gwen E. Gustafson Department of Psychology, U-1020, University of Connecticut, Storrs, CT
06268, USA, gwen.gustafson@uconn.edu

Cindy Harmon-Jones Department of Psychology, Texas A&M University, College Station,
TX, USA, chjones@psych.tamu.edu

Eddie Harmon-Jones Department of Psychology, Texas A&M University, College Station, TX,
USA, eddiehj@gmail.com

Bridgette D. Harper Department of Psychology, Auburn University at Montgomery, Montgomery, AL, USA, bharper3@aum.edu

Julie A. Hubbard University of Delaware, Newark, DE 19716, USA, jhubbard@psych.udel.edu

Carroll E. Izard University of Delaware, Newark, DE 19716, USA, izard@psych.udel.edu

Zoltán Kövecses Department of American Studies, Eötvös Loránd University, Budapest, Hungary, zkovecses@ludens.elte.hu

Elizabeth A. Lemerise Department of Psychology, Western Kentucky University, Bowling Green, KY 42101-1030, USA, elizabeth.lemerise@wku.edu

Jennifer S. Lerner Harvard University, Cambridge, MA 02138, USA, jennifer_lerner@harvard.edu

Michael Lewis University of Medicine and Dentistry of New Jersey, Newark, NJ, USA, lewis@umdnj.edu

Paul M. Litvak Carnegie Mellon University, Porter Hall, PA 15213, USA, plitvak@andrew.cmu.edu

David Matsumoto San Francisco State University, San Francisco, CA, USA, dm@sfsu.edu

Meghan D. McAuliffe University of Delaware, Newark, DE 19716, USA, meghanmc@psych.udel.edu

Amber McEachern Wichita State University, Wichita, KS, USA, iamemo@hotmail.com

Michael T. Morrow University of Delaware, Newark, DE 19716, USA, morrowm@psych.udel.edu

Raymond W. Novaco University of California, Irvine, CA, USA, rwnovaco@uci.edu

Roger Petersen Massachusetts Institute of Technology, Boston, MA, USA, rpeters@mit.edu

Carly K. Peterson Department of Psychology, Texas A&M University, College Station, TX, USA, carlypeterson@gmail.com

Michael Potegal University of Minnesota, Minneapolis, MN, USA, poteg001@umn.edu

Peihua Qiu University of Minnesota, Minneapolis, MN, USA, qiu@stat.umn.edu

Eric C. Reheiser University of South Florida, Tampa, FL, USA, reheiser@cas.usf.edu

Martin Reuter Department of Psychology, University of Bonn, Bonn, Germany, martin.reuter@uni-bonn-diff.de

Lydia J. Romano University of Delaware, Newark, DE 19716, USA, lromano@psych.udel.edu

Klaus R. Scherer Department of Psychology; Swiss National Center for Affective Sciences, University of Geneva, Geneva, Switzerland, klaus.scherer@unige.ch

Scott Schieman University of Toronto, Toronto, ON, Canada, scott.schieman@utoronto.ca

Lynn Schrepferman Wichita State University, Wichita, KS, USA, lynn.schrepferman@wichita.edu

David Schultz Department of Psychology, University of Maryland – Baltimore County, Baltimore, MD 21250, USA, dschultz@umbc.edu

Katherine Shonk Harvard University, Cambridge, MA 02138, USA, kshonk@hbs.edu

James Snyder Wichita State University, Wichita, KS, USA, james.snyder@wichita.edu

Charles D. Spielberger University of South Florida, Tampa, FL, USA, spielber@cas.usf.edu

Gerhard Stemmler Faculty of Psychology, Philipps-Universität, Warburg, Germany, gerhardstemmler@online.de

Larissa Z. Tiedens Stanford University, Palo Alto, CA 94304, USA, tiedens_larissa@gsb.stanford.edu

Thomas M. Tripp Washington State University at Vancouver, Vancouver, WA, USA, ttripp@vancouver.wsu.edu

Gerben A. Van Kleef Department of Social Psychology, University of Amsterdam, Amsterdam, The Netherlands, g.a.vankleef@uva.nl

Ajay Wasan Departments of Anesthesia & Psychiatry, Harvard Medical School, Brigham & Women's Hospital, Boston, MA, USA, awasan@partners.org

Pamela G. Whitney Department of Psychology, U-1020, University of Connecticut, Storrs, CT 06268, USA, pamela.whitney@uconn.edu

Janice Williams University of North Carolina, Chapel Hill, NC, USA; LaGrange, GA, USA, jwill22@bellsouth.net

Janet Wingrove Institute of Psychiatry, King's College London, London, UK, j.wingrove@iop.kcl.ac.uk

Tanja Wranik Department of Psychology; Swiss National Center for Affective Sciences, University of Geneva, Geneva, Switzerland, tanja.wranik@unige.ch

Seung Hee Yoo Yale University, New Haven, CT, USA, seunghee.yoo@yale.edu

Sarah Zukerman Massachusetts Institute of Technology, Boston, MA, USA, zukerman@mit.edu

About the Editors

Michael Potegal, Ph.D., L.P. is an associate professor of pediatrics and neurology at the University of Minnesota Medical School. He received his Ph.D. in physiological psychology from MIT in 1969 and held postdoctoral fellowships in neurophysiology at the Columbia University College of Physicians and Surgeons and the University of Amsterdam (the Netherlands). Prior positions include research scientist at the N.Y. State Psychiatric Institute and NRC Senior Resident Research Associate in the Walter Reed Army Institute of Research. Prof. Potegal has served as program chair (1986) and host (2006) of the biennial meetings of International Society for Research on Aggression (ISRA), as ISRA's representative at the UN, as ISRA council member, and as chair of the Young Investigators Program.

In 1993, Prof. Potegal integrated research and clinical interests by clinical training as a pediatric neuropsychologist, first at the University of Wisconsin and then at the University of Minnesota. He has received numerous university, foundation, federal, and international grant awards, has participated in NICHD review groups, and has published 75 papers and edited 3 books. After early studies on egocentric localization and vestibulo-spatial functions of the caudate nucleus, his research has focused on the time course and neural mechanisms of anger and aggression, most recently in children's tantrums. Recurrent themes in Prof. Potegal's research include the internal mechanisms that drive external aggressive behaviors and the cross-species similarity of these mechanisms in humans and other animals.

Gerhard Stemmler, Ph.D. earned his degree in 1984 at the University of Hamburg. After professorships at the University of Freiburg, he became full professor of personality psychology at the University of Marburg (Germany) in 1994. Prof. Stemmler has served as dean of the Faculty of Psychology (1995–1996, 2004–2006), president of the German Psychophysiological Society (1996–1997), and speaker of the Section for Personality Psychology and Psychological Assessment in the German Society of Psychology (2002–2004). His studies of the psychobiology of emotion and personality have identified physiological influences on self-reported experiences and observed behaviors using approaches involving genetic polymorphisms, somatovisceral psychophysiology, and EEG characteristics. In the emotion domain, his work addresses psychophysiological specificity (e.g., anger vs. fear; expectancy-wanting vs. warmth-liking) and strategies of emotion regulation. He has also done work on conflicts in motivational behavior tendencies. Within the personality domain, Prof Stemmler's recent research addresses agentic extraversion and the effects of dopaminergic neurotransmission on behavior facilitation, positive emotionality, and working memory.

Charles D. Spielberger, Ph.D., ABPP is a distinguished research professor of psychology and director of the Center for Research in Behavioral Medicine and Health Psychology at the University of South Florida, where he has been on faculty since 1972. He previously directed the USF Doctoral Program in clinical psychology and was a tenured faculty member at Duke (1955–1962), Vanderbilt (1963–1966), and Florida State University (1967–1972). Prof. Spielberger is author, co-author, or

editor of more than 400 professional publications, including 6 books on the theory and evaluation of anxiety in sports, education, and across cultures. His *State-Trait Anger EXpression Inventory* (1988, 1999) is widely used while his *State-Trait Anxiety Inventory* (1970, 1983) is a standard international measure with translations in 72 languages and dialects. He is also the author of anxiety inventories and surveys for children, tests, and job stress. His research contributions have been recognized in awards from the American Psychological Association (1993), the APA Divisions of Clinical, Community, and International Psychology, the Florida Psychological Association (1977, 1988), the Society for Personality Assessment (1990), and the Lifetime Achievement Award of the International STress and Anxiety Research Society (STAR, 1998). Prof. Spielberger served as president of the American Psychological Association (1991–1992), the International Association of Applied Psychology (1998–2002), and the International Stress Management Association (1992–2000), as well as six other professional psychology organizations and four APA divisions. He has chaired the National Council of Scientific Society Presidents (1996–1997) and the International Psychology Committee of the National Academy of Science (1996–2000) as well as five APA national committees. Prof. Spielbergers’s current research focuses on curiosity, anxiety, depression, and the experience, expression, and control of anger; job stress and stress management; and the effects of stress, emotions, and lifestyle factors on the etiology and progression of hypertension, cardiovascular disorders, and cancer.

Part I

Introduction

Chapter 1

Cross-Disciplinary Views of Anger: Consensus and Controversy

Michael Potegal and Gerhard Stemmler

Abstract The chapters of this book review current research on anger across academic disciplines, including affective neuroscience, business administration, epidemiology, health science, linguistics, political science, psychology, psychophysiology, and sociology. The opening chapters address biological bases of anger; subsequent contributions consider its constituent and concomitant psychological processes. The last chapters address anger in social context. Cross-citations in each chapter highlight similarities and differences in viewpoint among investigators in different disciplines.

The chapters of this book review state-of-the-art research on anger across a number of academic disciplines, including affective neuroscience, business administration, epidemiology, health science, linguistics, political science, psychology, psychophysiology, and sociology. The opening chapters address the biological bases of anger, examining theory and scrutinizing experimental methodology. Subsequent contributions consider constituent and concomitant psychological processes and consequences of anger. Chapters in the last sections address anger in social context. Cross-citations in each chapter refer the reader to allied material elsewhere in the book. These connections are intended to highlight similarities among, and linkages between, the problems and ideas being addressed by investigators in different disciplines using disparate terminologies and citing non-overlapping sources (cf., Wranik & Scherer vs. Tripp & Bies). Across this spectrum of disciplinary perspectives there is satisfyingly great progress toward scientific consensus, but sufficient controversy to invigorate our research efforts and enliven our discourse.

1.1 Triggers and Targets, Functions, and Social Significance of Anger

Novaco notes that the recurrent thwarting of physical, emotional, and interpersonal needs that occur in the custodial care of mentally retarded individuals contributes to their heightened propensity for anger. More generally, there is a quite broad agreement that typical triggers of anger include frustration; threats to autonomy, authority, or reputation; disrespect and insult; norm or rule violation; and a sense of injustice. Some authors subsume these various provocations under a rubric of goal blockage. There is also a general agreement that the expression of anger very much depends on target and

M. Potegal (✉)
University of Minnesota, Minneapolis, MN, USA
e-mail: poteg001@umn.edu

social circumstance, governed by display rules that are learned in childhood (Lemerise & Harper, Hubbard, et al.). Wranik and Scherer uniquely focus on the unusual characteristics of anger at the self, which occurs with relative frequency.

The idea that anger in its most typical context serves to reinforce hierarchical social structure is given credence by some. The view that it functions to forestall transgression against the individual and prevent her/his subordination is more widely shared among our authors (e.g., Stemmler). Fessler in particular argues that the “male flash of anger” evolved as an important adaptation which sidesteps time-consuming conscious deliberation and vacillation and more-or-less automatically commits individuals to immediate, risky, but potentially reputation- and/or life saving reactions to challenge. Novaco lists the attention and motivation enhancing actions of anger, Litvak et al. document its energizing and optimism-mobilizing effects, and Lewis argues for its utility in problem solving. A number of authors cite Averill’s well-known finding that anger can help motivate constructive social interaction. The historical controversy about the beneficial vs. the baleful effects of anger continues in these pages, contrasting the foregoing views with a broad consensus about the widespread, mostly adverse and sometimes lethal effects of anger on individual health (Fernandez & Wasan, Williams), in the struggles of daily life (Scheiman), harsh child-rearing and adverse developmental outcomes (Snyder et al.), peer rejection and victimization (Lemerise & Harper), intimate partner discord (Dutton), disrupted negotiation (Van Kleef), workplace rumination and revenge (Tripp & Bies), assaults and injuries in psychiatric facilities (Novaco), and political strife and warfare (Petersen & Zukerman). Accordingly, Fernandez reviews psychotherapeutic approaches to helping people manage their anger.

1.2 Constituents of Anger

Most authors hold that anger, like other emotions, consists of distinct components with variable degrees of situation-dependent coupling among them. These components include patterns of peripheral physiological responses and brain activation, physical sensations, subjective feelings and experience, cognitions, and action tendencies. The intensely experienced physical sensations and subjective feelings associated with anger (Tripp & Bies) are captured in metaphors of a hot liquid under pressure that occur in many languages (Koveces); there is a notable congruence between these linguistic metaphors and the distinctive autonomic physiology of anger that involves increased blood pressure, total peripheral resistance, and facial warming (Stemmler). While Lewis argues that, by definition, anger must involve adaptive, problem-solving responses that can be seen as early as 2 months of age, perhaps, many authors share the view that the evolutionarily derived, ontogenetically primitive action tendency associated with anger is aggression (e.g., Berkowitz, Fessler, Litvak et al., Potegal & Qui). Neurophysiologically, Harmon-Jones et al. note that the differential activation of left frontal cortex that is characteristic of anger is maximized when there is a possibility of angry action.

Nonetheless, most authors distinguish sharply between anger and aggression at a theoretical level. It is certainly possible to be aggressive without being angry; this is aggression in its “proactive” form (Hubbard et al.). Conversely, anger without overt aggression is the norm in many cultures; the other, more prosocial responses to anger typical of well-enculturated adults are taught and learned during early social development. In psychological studies of emotional states, anger has been isolated from aggression. The situation is different with regard to trait anger, however, which was so strongly

correlated with aggression in Spielberger's questionnaire studies that it defined an Anger–Hostility–Aggression (AHA) complex. In some fields in which the bases of behavioral traits are investigated, such as genetics (Reuter) and neurochemistry (Bond & Wingrove), correlations of biological variables are almost exclusively to AHA rather than to anger per se. AHA in adults may correspond to “reactive” aggression in children (Hubbard et al.)

1.3 Anger – Quantity, Quality, and Time Course

Most authors agree that anger ranges along a dimension of intensity, from frustration and annoyance to rage. Dutton and colleagues distinguished between “subanger” (frustration, annoyance, and irritation) and anger. As reported by Scheiman, sociological studies have compared the demographic prevalence of three levels of anger: feeling annoyed, feeling angry, and shouting. Potegal and Qiu propose to quantify the intensity range using a statistical model that is based strictly upon observed behaviors. Anger clearly varies in intensity within an episode. At extreme intensity, people become swept up in their anger, do things they might not otherwise do, and experience these acts as at least partially involuntary. Fessler argues that it is exactly this potential that makes anger so effective a social threat. Potegal and Stemmler suggest that actions committed in such “blind rages” involve not just a disinhibition of aggression, but a capture of decision making by orbitofrontal cortex and related limbic structures which replace the usual control by more dorsal regions and thereby generate the seemingly ego-alien quality of these experiences.

With regard to time course, a number of authors consider rumination, which can prolong anger well beyond the triggering incident. Potegal (Chapter 22) and Novaco (Chapter 27) both address the legal implications of persisting anger. Litvak et al. point out the sometimes pervasive and persisting effects of anger, noting that following a national crisis, it can become a national mood. Potegal sketches the rise and fall of anger, addressing mechanisms of escalation and metaphoric processes of decay, quenching, and the still controversial notion of catharsis. Fernandez incorporates a form of anger catharsis into his treatment for anger. Petersen & Zukerman introduce a framework for political scientists showing how anger supersedes rational economic choice and plays into violent, tit-for-tat struggles of dominance and revenge within and between communities. They suggest the hypothetical processes of anger decline as possible guides to appropriate political intervention in, e.g., La Violencia in Columbia.

In contrast to the strictly unidimensional view, Lewis argues that rage is a specific response to shame which is qualitatively different from anger in being more intense, prolonged, and diffuse. Wranik and Scherer propose that there are many different shades and blends of anger and other emotions depending upon person and circumstance. In particular, they differentiate among constructive, malicious, and fractious action tendencies. Potegal and Novaco note the historical recurrence of the idea that there are separate “good” and “bad” forms of anger. Fessler suggests that moral outrage is a particular form of anger that functions to discourage social deviance.

1.4 Valence and Motivation

Anger is generally held to be a negative (aversive) emotion, but one that involves active approach, in contrast to the negative emotions of sadness and fear which involve inhibition and withdrawal, respectively. While anger itself is generally negative, it can be accompanied by positive feelings, such

as increased alertness, strength, confidence, determination, and pride (Harmon-Jones et al.; Litvak et al.). These observations are consistent with the idea of anger as an approach-related emotion. Litvak et al. demonstrate that anger increases the taking of risks and optimism about the outcomes of that risk taking. These authors also hypothesize that anger is retrospectively unpleasant (when one looks back its source), but prospectively pleasant (when one contemplates future actions motivated by it).

Hemispheric asymmetries in the cerebral mediation of emotion are well established. Alternative views in which the left hemisphere is associated with positive emotion, or approach, and the right with negative emotion, or withdrawal, have focused on anger as test case of an emotion which is negative but often involves approach. Harmon-Jones et al. conclude that left frontal activation is associated with anger-motivated approach but right frontal activation is associated with anger-motivated withdrawal. Behaviorally, anger-motivated withdrawal appears as running away in children's tantrums (Potegal & Qiu) and psychological distancing in adults (Novaco.)

1.5 Information Processing, Appraisal, and Blame

Even more than other emotions, state and trait anger has been shown to narrow and direct attention, skew information processing, and bias judgment of both expressers and perceivers (Litvak et al.; Schultze et al.) Neurologically, a number of authors cite the role of the amygdala in the perception of threateningly angry faces. Wranik and Scherer have contributed to and present a prevailing view (cf., Litvak et al.; Tripp & Bies) that a stagewise series of appraisal processes are intrinsic to the elicitation and experience of emotion, recursively proceeding and accompanying its evocation. They propose that appraisals occur on levels from the sensorimotor through the propositional, and are often automatic, rapid, and unconscious (especially if the anger is a well-practiced response to recurring situations, as in many familial conflicts.) Berkowitz asserts several important theoretical and experimental caveats to the notion that such "top-down" appraisal processes are necessary for the emotion of anger in particular. He argues that appraisal does not satisfactorily account for such "bottom-up" phenomena such as the augmentation of anger by somatosensory feedback from gesture, vocalization, and facial expression. In his alternative neo-associationistic model, activation of one or another node of an anger network by insult, discomfort, or other aversive stimuli will activate other nodes in the network, e.g., impulses to action, like aggression (cf., Litvak et al.). Potegal and Stemmler propose the intermediate position that processing along known neurological pathways constitutes obligatory appraisals of certain sorts, but that, e.g., evocation of memories within the temporal lobe might have a neo-associationistic cast.

The question of blame is a particular issue. One of the sequentially activated components of Wranik and Scherer's appraisal model is an assessment of who is responsible for the offending event, and whether their action was intentional. (Tripp & Bies distinguish between bad-enough self-ish intent and more egregious malevolent intent.) Berkowitz cites evidence that while blame may commonly precede anger, it is neither necessary nor sufficient. In some cases it may be, quite literally, an afterthought; we become angry first and then search for someone or something to blame our anger upon. Berkowitz offers the case of pain-elicited anger as a counterargument to the necessity for cognitive appraisals that include blame. However, Fernandez and Wasan argue that acute pain involves a necessary, if brief and unconscious, attribution of blame, perhaps even the personified pain itself. In chronic pain, rumination leads the individual to blame, e.g., the person responsible for the injury/illness, the medical healthcare provider, insurance carrier, employer, significant other, self, and/or God.

1.6 Development, Gender, Personality, and Psychopathology

Litvak et al. note that anger has such potential social impact that even 10-week olds are equipped to distinguish it from other negative emotions. Authors agree that the expression of anger appears in the first year of life, can play an important negative role in parent–child interactions (Snyder et al.) and peer relations (Hubbard et al.) and becomes socialized for most children into more acceptable forms during development by appropriate parenting; children who cannot learn to self-regulate their anger are at risk for externalizing psychopathology (Lemerise & Harper). Although there appear to be few gender differences in the experience of anger (as opposed to the experience of other emotions), by 4 or 5 years of age, girls are more likely to mask anger, and to cry, and are less likely to be physically aggressive, than boys (Fischer & Evers, Scheiman). Some authors suppose that these differences are determined by gender differences in size and strength and by women’s fear of disrupted relationships and social opprobrium; but Fessler argues that it is mostly due to the biological necessity for mothers to protect their own survival in order to raise their children to the point where they can pass their genes into the next generation. There is substantial agreement that trait anger varies across individuals (Spielberger and Reheiser), that it has biological roots (Reuter; Bond & Wingrove), and that some of the physiological and psychological characteristics of anger are seen most clearly, or sometimes only in, high trait anger individuals (Schultz et al.). Wranik and Scherer suggest that individual differences in trait anger may be logically traceable to differences in appraisal bias. What may be adaptive as a brief flash becomes psychopathological as an unremitting glare; Novaco’s review highlights the accentuation of anger in dementia and depression; its prominence in PTSD as well as in several personality disorders, and its centrality in Intermittent Explosive Disorder.

1.7 Other Emotions

Most authors distinguish anger from other emotions, in keeping with one or another version of Differential Emotions Theory. Anger is often associated or blended with other strong emotions, such as fear (Snyder et al.; Tripp & Bies) or sadness (“distress” Potegal & Qiu); according to the clinical literature, it has an especially powerful interaction with shame. The general distinction among emotions is based upon decades of painstakingly accumulated and exhaustively analyzed evidence for differences in their facial (Matsumoto et al.) and vocal (Green et al.) expression as well as their somatovisceral physiology (Stemmler). Evidence for differences in the neural substrates of various emotions has begun to emerge much more recently (Potegal & Stemmler), although Wranik and Scherer raise some questions about the specificity of these behavioral and physiological observations. Working with quite different sorts of data, Matsumoto et al., Green et al., and Kövecses express the consensus that anger and other emotions are broadly similar across cultures, although there can be notable local, cultural modifications of the expression and perception of these presumably biologically based universals.

Variations of, and alternatives to Differential Emotions Theory, e.g., that anger and other emotions refer to prototypes or scripts for feelings and behavior or families or fuzzy hierarchies of emotion, are noted by several authors. Some authors note, and Green et al. describe in detail, an alternative to the differential emotions view in which “core affects” (pleasure or displeasure) are the basis upon which experiences of emotion are socially constructed. Supporters of this perspective tend to downplay the substantial and still emerging evidence for biological rootedness and physiological specificity of anger. Time will tell which model(s) invigorate the most productive research and yield the most insightful results.

Chapter 2

A Brief History of Anger

Michael Potegal and Raymond W. Novaco

Abstract Stories, myths, and religious beliefs reveal the powerful role that anger has played in human affairs since the beginning of recorded history. The projections of anger into the supernatural by ancient and pre-literate societies trying to account for the terrifying vagaries of nature testify to their experience with, and appreciation of, the baleful influence of anger in the human sphere. It has served as an instrument of the moral order, as cast in religious narratives and works of art, literature, and drama, and as legitimized in social rules. Various philosophies of human nature, moral conduct, and the search for perfection in human behavior have struggled to determine the essentials of anger. It is fundamentally linked to our representations of personal and societal order and disorder.

Stories, myths, and religious beliefs reveal the powerful role that anger has played in human affairs since the beginning of recorded history. The projections of anger into the supernatural by ancient and preliterate societies trying to account for the terrifying vagaries of nature testifies to their experience with, and appreciation of, the baleful influence of anger in the human sphere. It has served as an instrument of the moral order, as cast in religious narratives and works of art, literature, and drama, and as legitimized in social rules. Various philosophies of human nature, moral conduct, and seeking of perfection in human behavior have struggled to determine the essentials of anger. It is fundamentally linked to our representations of personal and societal order and disorder. Woven into the historical tapestry of human affairs are certain recurrently thematic ideas about anger. We present some of these here.

2.1 Anger: Supernatural and Superhuman

One indicator of the recurrent concern with anger is its projection onto animistic ghosts, spirits, and demons; the gods of the polytheistic pantheons; and the divinities of the modern monotheisms. The specific attributes of these projections served to illuminate anger's effects on social organization and life. Other indicators include extensive lexicons for anger as well as cultural interdictions against it.

The gods must be angry. In many preliterate, animist cultures, angry, malevolent spirits were (and are) believed to cause misfortune. Some were modeled on dangerous wildlife. In the pre-Hispanic

M. Potegal (✉)
University of Minnesota, Minneapolis, MN, USA
e-mail: poteg001@umn.edu

American Southwest, where water was rare and precious, Pueblo Indians believed that intrusion into springs inhabited by horned serpent spirits would provoke these irascible entities into retaliation by drought or flood (Phillips, VanPool, & VanPool, 2006). Other spirits are more anthropomorphic. As one of the innumerable examples, among the Celts, sudden shooting pains localized to a particular area of the body with no visible cause were “*elfshot*,” the pain of a magical arrow shot by angry elves (Froud & Lee, 1978). In Korea, ghosts are not entitled to *chesa* (feast food), whether they are ancestors who haunt their own households or wandering strangers. Always hungry and full of *han* (resentment and sorrow at having died unsatisfied), they are held responsible for sickness and injury suffered by individuals and households (Kendall, 1985).

The anger of the gods is more dangerous than the anger of the spirits. Anthropomorphically jealous or vengeful gods are found in a number of elaborated mythologies. Was it that early people could only conceive of gods with human characteristics? Alternatively or additionally, the projection of human emotions into supernatural beings served as explanation for frightening, uncontrollable, and otherwise inexplicable, social and natural disasters, especially in cultures where both fortune and misfortune imply agency. In Assyrian cuneiform accounts (circa 1200 BCE), their conquest of Babylonian cities meant that those cities had been abandoned by their guardian deities; the messengers of the departing gods were demons who brought wind, plague, and other calamities (Buchan, 1980). When the east coast of Sri Lanka becomes excessively hot, bringing drought and disease, the goddess Pattini must be angry (Leslie, 1998). Floods of China’s Yellow River were attributed to the anger of the river god, Ho Po (Lai, 1990). Aegir, the Norse ocean god, caused storms with his anger. Thunder and lightning were attributed to the anger of the original Zeus of Greece, Thor in the Norse pantheon, and the Mayan Chac. Shango, the Nigerian Yoruba god of thunder and war, carries a lightning spear. He is still worshipped in the African-derived cults of Central America and Brazil and in the Santeria religion of Cuba (Wescott & Morton-Williams, 1962).

Specific words in ancient Hebrew (*’anaph*) and Greek (*Mēnis*) distinguished the power of divine anger, “dreadful, often fatal . . . to be feared and avoided” (Considine, 1986), from merely human irk. In certain versions or stages of the elaborated polytheistic mythologies, anger itself becomes personified as a deity. In Zoroastrianism, a religion dating at least to ninth/tenth century BCE, Aeshma (*Aēšma*, he of the bloody mace) is the demon of wrath. In Hindu mythology, Manyu is one of the 12 aspects of Siva who was himself born of the anger of Brahma. In the Greek parsing of the varieties of anger, Nemesis was the goddess of righteous anger and divine retribution against those guilty of hubris, i.e., the fault of assuming god-like characteristics. The three female Erinyes, supernatural personifications of the vengeful anger of the dead, become the Roman Furies. Lyssa was the goddess of insane rage in the Greek pantheon; Ira is her Roman equivalent. Rabies derives from the Latin *rabere* (madness), the virus that makes dogs *rabid* was designated a genus of Lyssavirus.

What the gods were angry about. The polytheistic deities had recognizable human motivations. Poseidon felt himself the equal of Zeus and was angered by his brother’s power over the Olympians. However, what wrathful gods were most often wrathful about was disloyalty (worship of other gods) and disobedience (failure to observe rituals and prohibitions). Some insight into the nature of that projection is that anger about disloyalty/disobedience is especially characteristic of parents and chiefs. While community members may *experience* anger at the social deviance of others, *expressing* that anger is the particular province of dominant individuals and leaders who are deemed to be justified in doing so. Consider, for example, the role of chiefs’ *song* (justified anger) as moral guidance for Ifaluk islanders (Lutz, 1988) or the routine early morning moral instruction of younger band members by a Yanomami *pata* (leader/elder) whose angry shouts about their antisocial behaviors oblige them to placate him (Alés, 2000). Similarly, military training drill instructors are notorious for their (orchestrated) angry bark at the performance imperfections of recruits under their command.

The fearsomely angry personage featured in Greek and Roman stories is most often a monarch. The Roman emperor Caligula, infamous for his anger and insane murderousness, blended myth and terrifying reality when he declared himself a god (cf. Seneca, 44/1817).

Do people really believe in angry gods? The Airo-Pai, another Amazonian group, quiet their angry children by telling them that their shouts will attract *huati*, spirit monsters who feed on human souls (Belaunde, 2000). Where and when in history the anger of the gods was interpreted literally, metaphorically, or was used to frighten the children is unknown. Evidence for a literal interpretation comes from hundreds of sixth century and later Greco-Roman “curse tablets” in which people invoked various gods to wreak vengeance upon specific individuals for specific offenses (Harris, 2001). Stronger evidence is the widespread occurrence of appeasement rituals and ceremonies to abate supernatural anger ranging from the fourteenth century BCE Hittite appeasement prayers (Singer, 2002) to the still current offering of “cooling” rituals and foods to Sri Lanka’s Pattini. The hungry, angry ghosts of Korea are placated with occasional bits of food, and, when they become really noisome, by the raucous, female-dominated ritual of *kut*. Horses were sacrificed to Ho Po as late as the Han dynasty (206 BCE–220 CE, Lai, 1990). At least a fraction of the human sacrifices that were once prevalent in the Old World, infamously numerous in the New (Davies, 1981) and said to persist in isolated places today (Tierney, 1989), were meant to prevent or reverse natural disasters and appease the anger of the gods. The Aztecs, for example, sacrificed children to the rain god, Tlaloc – the children’s tears were thought to be a good omen (Duverger, 1983). For the affront of worshipping Baal, God says to Moses “Take all the chiefs of the people and impale them in the sun before the Lord, in order that the fierce anger of the Lord may turn away from Israel” (Numbers 25:4, New Revised Standard Version). If people did not fully believe in divine anger, they were concerned enough to hedge their bets. And, by implication, the gods not only become angry like humans, but they can be appeased like us, too.

Divine anger, one at a time. As the multiplicity of gods were banished from the Mid-East and Europe by the major monotheistic religions, deity gradually became less anthropomorphic, less sexualized, and more detached from the local landscape, distant, and abstract. Interestingly, however, Yahweh (later God) retained great capacity for anger. In the Older Testament (Hebrew Bible), Yahweh spent a lot of time being mad at people, individually and collectively. He banished Adam and Eve from the garden for disobedience and must have been really angry because he sentenced Adam and all his descendents to life at hard labor with no chance of parole. In anger, Yahweh unleashed the flood upon the world, destroyed Sodom and Gomorrah, visited plagues upon the Egyptians for not freeing the Israelites, then punished the Israelites for making the golden calf (Deut. 9:8,19,22) and on and on. In the Requiem mass (the Latin Mass of the Dead or Missa de Profundis), God’s anger is represented in the Dies Ire (Days of Rage) segment of the liturgy. So familiar is God’s anger that it is referred to as “the wrath” without further attribution.

In context and form, Yahweh’s anger is as it was for earlier gods. The sin of verbally disrespecting him has its own name, “blasphemy.” He punishes the Israelites as a father would punish errant family members; the avatars of his anger include wild beasts, famine, pestilence, and war. In a more nuanced interpretation of scripture, the destruction of the temple, devastating to the writers of the Bible because of its centrality to Jewish spiritual life, could not possibly be because the Babylonian gods were stronger than almighty Yahweh, but must be because he allowed it. Therefore, he must have been angry at the Jews for their disloyal and disobedient breaking of the covenant (worship me and you will have land and children). In the New Testament, God’s less frequent but still considerable anger is focused on disloyal non-believers (e.g., Rom. 10:14,17). Such beliefs persist, as indicated by the claims of a few Christian clergy that, e.g., the terrorist attacks of Sept. 11, 2001 or the flooding and deaths in New Orleans and other Gulf Coast cities following Hurricane Katrina in 2005 indicated God’s anger at American sins. The biblical future is as fraught as its past: On Judgment Day, 7 vials

of God's wrath (plagues) will be poured upon the antichrist, the wicked shall be resurrected in order to be burned in anger along with the earth, and so forth (Rev 16: 15–16).

The Bible is full of God's wrath, yet the disturbed and impulsive anger of everyday human experience is inconsistent with divine perfection. Some clerics argued that his anger did not involve revenge and that he was in control of his mind and emotions. Justification of God's anger as the divine version of "righteous" anger, discussed below, appears in various books of the Bible, such as Psalms, Zephaniah, and Ezekiel. Faced with this knotty theological problem, some Stoic philosophers and Christian ecclesiasts (e.g., Augustine) simply denied that God could be angry.

The Qur'an is replete with talk of rejection, slander, ridicule, curses, threats, punishment, torment, fighting, killing, and the burning of unbelievers in hellfire, at least in English translation. Anger itself is remarkably rare. The Al-Fatiha Surah (opening chapter) is recited five times a day by devout Muslims who ask to be shown the "... straight path, not the path of those who have earned Allah's anger" (Qur'an 1:1¹). His "Wrath upon Wrath" at unbelievers appears in Surah 2:89–91. The wrath of the unbelievers themselves is noted a few times [in Surah 3:119 they bite (off) their fingertips in rage], as is Moses' anger at the Hebrew's worship of the golden calf. The notable paucity of anger in the Qur'an, in contrast to its abundance in the Judeo-Christian Bible, may reflect its common denial in Arab cultures (e.g., Somer & Saadon, 2000). However, in ninth century *hadith* literature, the Prophet's anger was key in acceptance into, or rejection from, the *umma* (community of believers, Ghazzal, 1998). After the 2005 earthquake in Pakistan killed more than 90,000 people, the mass media there promulgated the belief that the quake was Allah's punishment for sinful behavior. The mullahs incited followers to smash television sets, which had provoked Allah's anger and hence the earthquake.

When gods may be angry, but people may not. In some belief systems, anger may be a supernatural prerogative, to be expected of ghosts or gods but unacceptable for mortals. Cultural restrictions on expressing, or sometimes even experiencing, anger exist in many cultures around the world. These prohibitions generally emerge from three interrelated beliefs: (1) fear of social and/or physical reprisal and, especially in cultures where all harm is thought to result from the malicious action of others, or retaliation by witchcraft; (2) harboring anger invites ill-health and misfortune; and (3) expressing anger is inappropriate for adults; it is irrational, amoral, bestial and/or childish, and very shameful.

Despite the tensions, animosities, and suspicions of malevolent intent that are commonplace in band and village life, people in these groups are careful to maintain cordial social relations. Multiple anthropological accounts attest to these beliefs in Tahiti where they are a part of general pattern of harm avoidance (e.g., Levy, 1973). Variants are found throughout Indonesia and Malaysia, e.g., Java and Bali (Hollan, 1988). Among the Kenyan Taita, unwitting but dangerous "anger in the heart," engendered by violations of a person's jural or kinship rights and revealed by divination, was expelled in the *Kutasa* ritual by drinking and then spitting out beer (for men) or cane juice (for women); consuming the animal sacrifice that followed restored peace and harmony and brought the blessings of children and well-being (Harris, 1978). Notable examples also occur among indigenous people of the New World. The small number of Inuit in the Utku band belies their renown as exemplars of people who do not show anger even in situations that would outrage others; they describe the angry behavior of foreigners as "childish" (Briggs, 1970). Their ultimate sanction of ostracism is potentially fatal in a group whose members must cooperate for survival in an extremely hostile environment. The highland Maya of Mexico similarly maintain an ideal of cool individual non-reactivity and non-confrontational interpersonal relations based on the threat of "buried" anger of the "fevered heart" (Groark, 2008). For the Airo-Pai, as for other groups living along the Amazon, anger is taken as a sign of aggression, and its display is deemed a most significant offense against the community (Overing & Passes, 2000). The Airo-Pai believe that the angry individual loses all moral sense, treats

kinfolk as prey or enemy, and is no longer in a human state. Anger leads to sorcery; e.g., it invites the *huati* to mislead the individual into hunting other people like they were forest game. The Airo-Pai remind themselves aloud that anger against kinfolk is vain and purposeless and that one ought not be angry. Conversely, mastery of anger represents a successful transformation of inimical spirits (Belaunde, 2000). Historically, each time the Delaware Indians were displaced westward by white settlers, they became concerned about *kwulakan*, a taboo against anger instituted to prevent divine retribution (Miller, 1975). In the American Southwest, during the year-long preparation for the winter solstice festival, *Shalako*, the members of the Zuni priesthood of “sacred clown” impersonators must refrain from all negative emotions, including anger, lest it disturb these most dangerous of deities (Tedlock, 1983).

From whence comes the belief that anger may be expected of gods, but is unacceptable for people? The Airo-Pai fear the anger of infants whose cries may enrage their parents to the point of infanticide; their creation myths contain just such an event (Belaunde, 2000). What the Yanomami *patas* harangue about are antisocial behaviors, such as theft from gardens and propositioning married women, which can engender anger, group fissioning, and/or homicide (Alés, 2000). Perhaps, even before the rise of the state and organized religion, repeated experience in family groups and tribal life over the millennia generated a deep understanding that the divisive and disruptive effects of anger lead to social disorganization and intragroup aggression. This understanding is expressed in early taboos against anger. This may be especially true for collectivist cultures as opposed to individualist/egalitarian cultures. In any event, such prohibitions have subsequently been imposed from above by religious and other governing hierarchies with vested interests in stability and social control (a group’s healthy fear of divine anger tends to keep their priesthood in business).

The Greeks had many words for it. National (cultural) epics, based on oral traditions predating written history, provide the oldest depictions of human behavior extant. Albeit stylized by narrative conventions, they are rife with references to anger and its expression. In the oldest epic extant (circa 2700 BCE), when Gilgamesh, god-king of Sumer, rejects the advances of treacherous Ishtar, she angrily calls upon her father, the god Anu, to release the Bull of Heaven to destroy him. Rejection and insult trigger anger, which motivates indirect aggression. How modern!

The *Illiad* of Homer (circa eighth to seventh century BCE, trans 2004) begins with the phrase “Wrath (*Mēnis*) of Achilles.” Thus, the first word in the Western canon is anger! The use of “*Mēnis*” recognizes Achilles’ semi-divine parentage and the intensity of his anger. Anger in the *Illiad* is described in full-blown complexity; it arises from various social causes (e.g., insults to honor, killing of a friend), and its many manifestations include facial expressions (“blazing eyes,” frowns, tearing of hair, and so forth) and a panoply of verbal (complaints, insults, threats) and physical acts (aggression and homicide). Literary critics have regarded the *Illiad* as an extended meditation on anger. Most (2003, p. 54) notes “Agamemnon’s haughtily self-righteous fury, Achilles’ astonished indignation, sullen resentment, uncontrollable rage and glacial wrath . . . Theristes’ obstreperous defiance . . . Odysseus’ irate disdain . . . Helen’s partly relieved contempt . . . Apollo’s bland vengefulness . . . Aphrodite’s admonitory scorn . . . Ares’ insane ferocity . . .” All of these are perfectly recognizable to modern audiences. In contrast to later works of antiquity, characters in the *Illiad* moderate or master anger relatively rarely. Indeed, characters are criticized for showing anger insufficient to the provocation (Cairns, 2003). This is notably different from the millennium older epic whose central theme is the close bond of friendship that arises between Gilgamesh and the wild man Enkidu when he masters his own anger and declares his respect for the god-king.

The ancient Greek texts pose a challenge for the appropriate translation of many words connoting variations in the intensity, context, and meaning of anger; such multiplicity has been taken as one indicator of their pre-occupation with this emotion. Besides *Mēnis* and *Nemesis*, there is *chalepainō*

(annoyance), *kotos* (resentment), *cholos* (bitterness, literally “bile”), *thumos* (in the more general context of zeal or energy), *orgē* for intense anger, sometimes bordering on madness, and others. These words were at least partially overlapping in meaning, sometimes used interchangeably, and changed over time (cf., Harris, 2001). Note that even preliterate cultures have assortments of words for variations in anger (e.g., Briggs, 1970; Lutz, 1988). In comparing 47 non Indo-European languages that differed in the number of emotion categories that were labeled, Hupka, Lenton, and Hutchison (1999) found that anger, along with guilt, were the first categories of emotion to be labeled. However, different languages and cultures may have no exact equivalents of English emotion words, including “anger.” Among the Philippine Ilongot, *liget* indicates both anger and grief and is seen as providing the energy needed for, e.g., a retaliatory headhunting raid (note the similarity in connotation to the Greek *thumos*). Words in other cultures/languages may also be conditioned by the triggers for and motivation of the anger, social roles, and context (e.g., directed against kin or non-kin), classification as justified or unjustified, and so forth (e.g., Harkins & Wierzbicka, 2001; Myers, 1988).

Anger and manhood in other warrior cultures. In stark contrast to cultures that reject anger and aggression, in principle or in practice, are warrior cultures in which anger was cultivated as a special trance-like state that produced indifference to wounds and fearlessness in battle. The best known examples are the Norse or Viking “berserkers,” the armor-scorning fighters of myth and history whose rabid fury was described in Hall’s (1899) classic treatise on anger. The berserkers’ periodic insanity was perhaps enhanced or engendered by ingesting mushrooms, which would account for the psychotic features of their rages (including visual hallucinations, Fabing, 1956). Whatever the cause, the dangerousness of these fighters would dissuade kinsmen of individuals killed by a berserker from their culturally mandated revenge (Dunbar, Clark, & Hurst, 1995). Speidel’s (2002) extensive historical analysis traces mad, recklessly fighting, shape-shifting “true berserkers” from the second millennium BC, including Assyrians, Hittites, Thracians, Celts, tribes of Italic, Germanic, and Anglo people, and Aztecs. Various berserker groups fanned their fury with dances, a possible remnant of which is the Maori *haka* with its facial contortions, eye-bulging, tongue gyrations, body slapping, and grunts, all of which convey a wild and fearless, if stylized, ferocity.

“Wild man” and “amok” are related syndromes of highly systematized, eruptive, and frenzied violence in Micronesia and other Pacific Island societies. *Amok* in southeast Asia derives from a Malay word for a murderous frenzy with intense rage. Kon (1994) traced its origins on the Malay Archipelago in the mid-sixteenth century and its subsequent appearance in other societies, including a seventeenth century warrior class in southern India (cf. Spores, 1988). It is almost exclusively a male syndrome, suggesting that it may be a cultural exaggeration of the “male flash of anger” whose adaptive value Fessler discusses in this book. However, amok and allied phenomena are viewed as temporary insanity, even in these cultures (e.g., Carr & Tan, 1976; Gaw & Bernstein, 1992); as described in Novaco’s chapter, they can involve deep psychopathology and persisting psychosis.

2.2 The Philosophy and Psychology of Anger

As anger came under rational scrutiny, and was detached from the supernatural, competing interpretations emerged. Anger as a bestial passion rooted in biology can be contrasted with anger as integral to manhood and with anger as a motivator of just action. The metaphor of bestial passion has not been abandoned, but has been transformed into more modern views of anger in the context of development and gender, as well as in insanity, sin, or demonic possession. The view of anger as

integral to manhood and as in support of moral order also continues, as still seen in its role in the maintenance of social hierarchy.

The earliest literature. The Illiad's concerns with anger continued through the philosophies of classical Greek antiquity (fifth to fourth century BCE) and subsequent Greco-Roman commentary, drama, and oration. Why? Although fourth century Athens was famously rough and competitive, anger may have been no more rampant there than in, say, contemporary Washington, DC. Noting no evidence that Athenians went armed to the marketplace, Harris (2001) conjectures that this Greek preoccupation might have been due to the baleful effects of the anger of tyrants, as well as the disruptive consequences of anger on stability within, and competition among, their newly formed and relatively fragile city states. (For an empirically based view of the effects of anger in political context, see Chapter 32 by Petersen & Zukerman, this book.) In any event, the ideas introduced during that period continue to inform contemporary thinking and research. For example, Allen (2003, p. 79) translates Aristotle's definition of anger (*orgē*) as "...a desire, commingled with pain, to see someone punished, and which is provoked by an apparent slight to the angered person, or to something or someone that belongs to him, when that slight is not justified..." This definition includes injustice as a crucial trigger (e.g., Chapter 19 by Schieman and Chapter 24 by Tripp & Bies, this book), requires an appraisal of the situation (e.g., Chapter 17 by Litvak et al. and Chapter 15 by Wranik & Scherer, this book), and stipulates a response tendency of aggression (e.g., Chapter 16 by Berkowitz, Chapter 21 by Fessler, and Chapter 14 by Hubbard et al., this book).

Anger as bestial passion (and what to do about it). Anger is the prototype for the classical view of emotions as "passions" that seize the personality, disturb judgment, alter bodily conditions, and imperil social interaction. The "consensus theory," which developed in the centuries following the Roman era and held sway into medieval times, was that anger is the strongest of the "spirited" or "irascible" emotions whose function it is to obtain pleasure and avoid pain under conditions of difficulty (Kemp & Strongman, 1995). The theme of anger as a bestial passion or irascible emotion is followed logically by the theme that anger must be mastered by reason, will, and self-control. Lucius Seneca (44/1817), arguably the first scholar of anger, sought its eradication in the quest for tranquility of mind. So did the Roman Cicero before him and the Greek Plutarch after him. Earlier Greeks, such as Pythagoras, had taught that consciously restraining one's anger (refraining from speaking or acting when angry) would encourage temperance and self-control; this ancient prescription for anger management remains an element in current approaches (see Chapter 28 by Fernandez, this book). Pythagoras recommended music therapy, countering rage with melody. In Plato's Phaedrus, the charioteer of reason must master the wanton black horse of passion (trans. 1975); the verses of the Dhammapada, claimed to have been spoken by Buddha himself, contain a very similar metaphor (circa 500–400 BCE, Vernezze, 2008). In Freud's (1933) version, the rider is the ego who must control the horse, the id. The metaphor of "rider" above mastering "horse" below re-emerges, albeit fortuitously, in the contemporary neuroanatomical evidence for a balance of functional control, which varies reciprocally between the dorsal areas of lateral and medial frontal cortex that mediate cognition and executive control and the more ventral limbic structures that mediate emotion (e.g., Dolcos & McCarthy, 2006.)

The idea that anger is an irrational, even bestial, passion has a number of implications:

Anger as irrational/maladaptive. Although conflict and combat are fraught with provocations to anger that can motivate vigorous (or desperate) action, historical commentators have cautioned that anger is to be avoided because of its concomitant impairments in judgment. Seneca and others expressed the view that both in sport and in war, the disciplined combatants defeat the angry ones. The military strategy writings attributed to Sun Tzu (fourth century BC) depicted anger as a

fault upon which military commanders could capitalize. In his *Meditations*, Marcus Aurelius, second century Roman Emperor and a Stoic philosopher himself, wrote that yielding to anger was a sign of weakness. Likewise, postponing vengeance until one is calm is a frequently recurring admonition.

Development and gender. Although overt expression of anger is more typically associated with men, children and women were thought by some classical and medieval writers to be prone to excessive anger due to their lack of moral instruction, cognitive immaturity, or poorly developed rational faculties (Kemp & Strongman, 1995). The ancient Greeks believed that babies experienced anger from their first days (Hanson, 2003; see Chapter 11 by Lewis, this book for more modern views); Galen warned that anger was a precursor of severe disease in infants. As chronicled by Stearns & Stearns (1986), the first written use of “tantrums” is in British plays some years after 1748 where the term was used to belittle adult bouts of anger. As used by followers of Darwin through the 1860s, “tantrums” came to refer to children’s anger. In the West, such tantrums and other misbehaviors were commonly met with harsh and angry punishment, disciplinary tactics meant to “break the child’s will”; if he became enraged, he was punished further. Of course, such discipline taught children not to express anger to parents, but that anger and physical punishment were suitable responses to subordinates (such shaping of behavior is analyzed in detail by Snyder et al., this book (Chapter 29)).

In Classical Greek thought, men’s *orgē* was forthright, hot-blooded, and immediate while women’s *cholos* was weak, cold, and delayed (i.e., women schemed and were vengeful, Allen, 2003). Although some debate about the existence and nature of sex differences in anger remains, recent research generally indicates that women experience anger at least as intensely as men, and express it as least as often, but differ somewhat in the triggers and modes of expression (Chapter 20 by Fischer & Evers, Chapter 21 by Fessler, this book). Women are less likely to become physically aggressive, but more likely to cry and to express anger indirectly (through “relational” aggression). The Greek version of sex differences may have correctly identified these response elements, although their interpretation of these (and most other) social phenomena was relentlessly misogynistic. Whatever else it might be, women’s anger was always unacceptable. This tradition remains widespread. *Ngon* [speech] is the third of four culturally prescribed virtues for Vietnamese women, e.g., it means to speak softly and never raise the voice – particularly in front of the husband or his relatives (Rydstrom, 2003).

Insanity, sin, or demonic possession? In the Illiad, when Achilles learns of the death in battle of his close friend Patrocles, he is engulfed in a “black stormcloud of pain . . . (he) tore his hair with both hands” (p. 430), and he becomes “mad with rage” (Homer, 2004, p. 468). He viciously kills a prince of Troy, Hector, and defiles his body, unflinchingly dismissing Hector’s admonition that such defilement will anger the gods.

Orgē was regarded as a form of irrationality, illness, or insanity most notably by Herodotus, later by Galen, and by Seneca who endorsed the view of anger as a “short madness” (p. 222). Galen’s (1963) treatise on “passions and errors of the soul” frequently construes anger episodes as marked by madness, including the behavior of his personal friends, his mother, and the Roman Emperor Hadrian – “rage is a madness” (p. 42). As argued by Novaco (Chapter 27, this book), recent offshoots of this line of thought include (1) Fava’s (1998) “anger attacks,” which connote being seized by a pathological/disease entity that “explains” aggressive behavior and is then suitably “treated” by medication, (2) the general issue of the role of anger in psychopathology, and (3) the proposal that there should be specific diagnostic categories of anger (e.g., Kassino et al., 1995).

In another recent incarnation of anger as irrationality, it, like other “visceral factors” (Lowenstein, 2000), is seen to impair rational economic reckoning, in part by disrupting normal time discounting of reward value. A neuroanatomical basis for this psychological phenomenon is suggested by Potegal

& Stemmler (this book). In any case, the result is behavior that may appear, and be experienced as, out of the individual's control or, conversely, as anger that has seized control of the individual (anger as a "daimonic," Diamond, 1996). The legal interpretations and implications of anger as form of mental defect are noted by Wranik & Scherer (Chapter 15), by Novaco (Chapter 27), and by Potegal (Chapter 22), this book.

A main line of Buddhist thought is that anger is a moral "blemish" that must be eschewed at all times to attain the tranquility of enlightenment. Anger is also seen as a form of suffering that arises from appraisals that one has been insulted, hurt, defeated, or robbed by another. It can be remedied by "binding the mind" to dismiss these thoughts. Among the reasons for doing so are that we have doubtless offended against others in our past lives, and being offended by others in this life is no more than just desserts (Verneze, 2008).

Anger as a mortal sin was introduced to Christianity in Paul's letter to the Galicians (circa 50 CE) naming anger as the fourth of the seven deadly sins (*Galicians 5:19–21 NRSV*). The precepts in this letter, which were later to have such a large impact on Catholic belief and practice, had deep historical roots, e.g., in ideas found in Proverbs (6:16–19), some of which can be traced, in turn, to Egyptian writings as early as the second millennium BCE. In Dante's *Inferno* (1308 CE), the wrathful damned claw each other through eternity in the fifth circle of hell, a burning Stygian marsh (the sullenly angry wind up buried in the marsh). But anger was never the most important of sins [Paul does allow Christians to be briefly angry, but warns them "to not let the sun set upon their anger" (Eph. 4:26.)] After the twelfth century, however, humility and its concomitant of anger control were no longer required of Catholic saints (Stearns & Stearns, 1986). One could be both angry and holy. Since the eighteenth century (e.g., Bellers, 1702), some Protestant denominations have re-focused attention on the evils of anger.

Anger as part of demonic possession, as opposed to mental illness, appears in ancient Egyptian and Hebrew sources (Isaacs, 1987). In the European "Age of the Demonic" (1550–1650 ACE), possession became more prominent in Jewish as well as Christian communities; "diabolic distemper" (excessive anger) was one sign of being a witch. During the Salem, Massachusetts witchcraft episode in the late seventeenth century, Pastor Samuel Parrish wrote in his church book that "The Devil has been raised among us, and his Rage is vehement and terrible . . ." (Trask, 1975). Remarkably, reports of demonic possession continue to the present. In 24 observed or recalled exorcisms by clerics in Rome, Italy, and Berkeley, California, all "possessed" individuals showed facial expressions described as angry, hate-filled, or "distorted" and had furious outbursts in which they might attack religious objects (they also exhibited other, more dramatic, and bizarre behaviors, Isaacs, 1987; Ferracuti, Sacco, & Lazzari, 1996). Clearly, the "script" for being possessed involves displays of anger.

The endpoint of this line of reasoning remains the vexing issue of anger control, which has been addressed in disparate ways by Stoic philosophers, Psalmists, Scholastics, Mayans, philosophers of the Enlightenment, American colonists, Victorians, Existentialists, early North American psychology, Dr. Spock, and by psychodynamic and cognitive-behavioral therapists, to name a few. Interventions for problematic anger have progressed substantially from common language prescriptions throughout the ages and across cultures. As Fernandez (Chapter 28, this book) describes, contemporary therapeutic intervention is theoretically anchored, assessment driven, and evidence based. Meta-analyses of anger treatment have found medium to strong effect sizes (e.g., Beck & Fernandez, 1998; Del Vecchio & O'Leary, 2004; DiGuiseppe & Tafrate, 2003; Sukhodolsky, Kassino, & Gorman, 2004), indicating that approximately 75% of those receiving anger treatment improve, compared to controls. There is certainly a cause for optimism.

Personality and biology. The observation that some individuals are consistently anger-prone invites explanations in terms of personality. The oldest parsing of anger characteristics that we have

come across is a distinction, written in Egyptian hieroglyphics, between. . . “angry (like a) monkey” and “angry (like a) bull”: The angry monkey is loud and showy, but not dangerous; the angry bull is not as flamboyant, but is threatening and very dangerous (Goldwasser, 2005). In contemporary Arab Tunisia, one must beware the angry camel (Maleej, 2004). In the Aristotelian view, personality is shaped by environment and experience, as ironically illustrated by Achilles’ anger at the jibe that his chronic *cholos* was a result of having been “nursed on bile” (Hanson, 2003). The contrasting Platonic view of character as predetermined is a precursor to rooting trait anger in biology. This view is strengthened by the reification of anger in acute physiology. Thus, for example, biblical Hebrew terms for anger that refer to the nostril (‘ap) or involve hard breathing (‘anaph) capture this particular physiological aspect of anger (Harrison, 1979). Green et al. (Chapter 9, this book) present this view in modern dress as the idea of embodiment.

Hippocrates’ concept of four humors as the basis of physiology and medicine, later popularized by Galen as integrated patterns of physiology and physiognomy, dominated Western thought until the mid-nineteenth century. In humoral theory, the sharp-featured, anger-prone, “choleric” (from *cholos*) person is ambitious, energetic, and dominant in social exchange. The choleric pattern results from an excess of yellow bile, which also corresponds to fire (more exactly, excess heat, cf. Irwin, 1947) in the four element theory of matter. A similar personality profile in India’s Ayurvedic system results from an excess of *Pitta*, the fire-related one of the three “*Doshas*” or elemental forces. Mythophysiology aside, the choleric combination of psychological features presages the empirical identification of Type A personality (Chapter 25 by Williams, this book).

Aristotle’s association of anger with heating of the blood around the heart (Kemp & Stongman, 1995) was a little closer to a telltale organ system (cf., Chapter 7 by Stemmler and Chapter 10 by Kövecses, this book). The recent discoveries associating anger and Type A personality with cardiovascular disease (Chapter 25 by Williams, this book) is one of the few current ideas with little historical precedent. As illustrated in chapters by Potegal and Stemmler, and by Harmon-Jones et al., and Stemmler, modern methodology has increased the focus on the neural bases of anger and clarified its peripheral physiological signs.

Anger in support of moral order: Aristotle and afterward. As Plato is a main source for the view of anger as bestial passion, Aristotle is cited for the alternative, conditional view that “. . .anger at the right person, on the right occasion, in the right manner. . .” can be appropriate, virtuous and ethically justified (*Nicomachean Ethics*, Book 4, Chapter 5, 1126b5–10). The admonition, noted above, to postpone revenge until one is calm, has not been universally accepted. In classical Athens, where reputations were always at stake and insults had to be met forthrightly, orators routinely argued that justice should be meted out quickly, “in hot blood,” after the crime. A favorite trope was that the law itself was angry at the accused, and you, the spectator, should be angry at him as well (Allen, 2003). In this rhetorical flourish, the idea of justified anger becomes commingled with, if not equivalent to, justice itself.

Experimental demonstrations that anger increases optimism and risk taking that can, in turn, prompt corrective action are relatively recent (Chapter 15 by Wranik & Scherer and Chapter 17 by Litvak et al., this book). However, the idea that anger triggered by injustice to the self or others generates the zeal and discipline for constructive action (Kemp & Strongman, 1995) has historical roots that are wide and deep. The Ifaluk word *song* means anger that is justified by threats to moral order; it is the only socially acceptable form of anger in that culture (Lutz, 1988). The Exnet of Paraguay, who abjure anger and rarely express it against community members, find it acceptable for shamans to direct their anger against malevolent witches and evil spirits (Kidd, 2000). Anger is also acceptable in confronting missionaries and government officials for the common good. In Greece, historically, *nemesis* came to suggest the resentment associated with injustice, which could not be allowed to go unpunished. More recent examples of good works motivated by righteous anger

are numerous. At least according to American presidential folklore, Abraham Lincoln's hatred of slavery and anger at slave owners was part of his motivation for the American Civil War and for his emancipation of the slaves (Paludan, 2006). The role of anger in motivating collective action in, e.g., the feminist movement, has also been described (Hercus, 1999). An extreme form of this idea is that anger, and even rage, is a "daimonic" of pure and beneficial creative energy which will engender psychological and social disorder if chronically suppressed (Diamond, 1996).

However, "righteous anger" is not necessarily constructive and prosocial, but depends on who is getting angry, what they do about it, and who is telling the story. While some of the ancients held that anger facilitated courage, others held that it facilitated a pseudo-courage fit only for barbarians. When peasants revolted in the late middle ages, their anger was not seen as noble or justified, but likened to the rage of beasts (Freedman, 1998). Lincoln's anger was constructive, but that of more recent American presidents has not been (Baker, 2000). In Tsytarev and Grodnitzky's (1995) instructive account, organized crime hitmen reportedly work themselves into righteous anger by fabricating imaginary injustices perpetrated by an intended victim. The notion that righteous anger deserves special status recurs in the frequent re-invention of two types of anger, e.g., the Epicurean view of appropriate "natural" anger vs. immoderate "empty" anger (Harris, 2001), St. Thomas Aquinas' distinction between righteous anger in response to evil vs. sinful rage (Reid, 2006), and Ellis and Dryden's (1987) "appropriate" anger which motivates rational problem solving vs. "inappropriate" anger which engenders irrational thinking. In this book, Lewis distinguishes between anger, which is appropriately focused on removing a frustrating goal blockage, and rage, which is driven by shame and has no specific goal. Wrانik and Scherer contrast "constructive" anger to "malevolent" and "fractious" (venting) anger expression and Fessler proposes a specific, evolutionarily based moral outrage as a reaction to, and corrective of, social norm violations. However, the idea that righteous anger is a specific kind of emotion may confound anger per se with its trigger ("rational" anger may be most frequently about perceived injustice to others), intensity ("rational" anger may be milder), temperament (well-adjusted individuals may be better able to channel their anger into constructive, pro-social action than impulsive, poorly regulated individuals), and also audience (congruence with pre-ordained beliefs of observers).

2.3 The Historical Ubiquity of Anger and the Exercise of Social Control

The work of several groups of scholars suggests that social tendencies to moderate anger have emerged in different times and places. However, these tendencies are pitted against the self-sustaining role of anger in maintaining the status quo of social hierarchy in each time and place.

Then and now. Anger was seemingly indulged in with little censure or shame in Western epic times, but advice about restraint appears as early as Sappho's (circa 600 BCE) line "When *orgē* is spreading through your breast, it is best to keep your yapping tongue in check." This translation is by Harris (2001), who traces a thoughtfulness about anger (and other human actions) and trends toward self-control that emerged in Greece around fourth century BCE. Hyams (1998) reconstructs similar trends in twelfth to thirteenth century England, and Stearns and Stearns (1986) document even more recent trends. The latter authors argue that anger (and other emotions) was more openly displayed in pre-nineteenth century Western society, and elicited less social concern and prohibition, than they do currently. There were loud arguments in city streets, derogatory nicknames and traditional curses in the countryside, and little expectation that anger would or should be moderated. In European cultures, affronts to masculine honor had to be met with anger and counter-aggression, if a man was to avoid shame. Examples of this tradition include medieval and Renaissance vendettas

between families as well as dueling among members of the aristocracy and, later, the mercantile class. So powerful were these traditions that civil and religious authorities were unsuccessful in suppressing them despite continuing interdictions. Notable New World examples include litigious neighbors of seventeenth century New England and the fractious American frontier. Particular subcultures that continue to honor the “angry response to affront” include the German aristocracy and American southerners (e.g., Cohen, Nisbett, Bowdle, & Schwarz, 1994). Anger directed against outsiders, social deviates, and “others” remains widespread (contemporary American rates of anger are reviewed by Scheiman (Chapter 19), this book.)

Against this historical background, Stearns and Stearns (1986) describe a gradual but prolonged shift toward a greater awareness of and appreciation for the role of emotions in social life. Based on diaries, books of advice, marriage manuals, and the like over the last few centuries, these authors annotate an “emotionology” that placed greater emphasis on tolerance, consideration of others, and the gentler passions and conversely, an increasing disapproval of the unbridled expression of anger. Such ideas were part of the eighteenth century European Enlightenment, which emphasized reason, education, and literacy; new esteem for tolerance and ridicule of those who were easily angered. Protestant views emphasized the spiritual equality of women, and subsequent nineteenth and twentieth century sermons and pamphlets of various Protestant denominations attested to the evils of anger. New ideals of romantic love in marriage and kindness toward children appeared, emphasizing the need for restraint in the family, and for masters with servants, and making anger in the home seems inappropriate. With the industrial revolution came increases in market activities requiring civil interactions with strangers while increases in prosperity helped reduce tension in the home. New social classes included shopkeepers who needed to maintain cordial relations with customers. In nineteenth century Victorian views, home was a haven in a heartless world where anger was not to be allowed. A literature on the control of anger for spouses and parents advised the avoidance of conflict. Women should not feel anger and men should control it; not to do so was a flaw in moral character. Distinctions were made between restrained emotional world of adults and the unrestrained world of children; it was during this time that the word “tantrum” was introduced.

In addition to tracking historical trends in anger reduction, Stearns and Stearns (1986) raise the question of who expresses anger to whom. Among the Utku, anger and aggression are directed only to dogs; under the guise of “discipline” it is freely, frequently, and violently expressed to this underclass of creatures (Briggs, 1970). On Ifaluk (Lutz, 1988), *song* is shown by those of higher status to those of lower status (e.g., chiefs to villagers, adults to children); the latter are expected to experience the appropriately fearful emotion of *metagu*. Seneca and other classical writers took special note of anger directed against slaves (Kemp & Strongman, 1995). (Plato advised restraint on the grounds that it made them less inclined to work and more inclined to murder.) As crafts emerged in the middle ages, so did anger directed toward apprentices (see Rosenwein, 1998 for other accounts of medieval anger). Later, house servants were routinely subjected to abuse, as documented in their personal accounts (Stearns & Stearns, 1986). These are all historical instances of classes of social subordinates being subjected to the anger of dominant classes. Although adult male anger directed toward women and children clearly predates any victimization in conjunction with such social classes, economic analyses can be applied to these situations as well.

Anger and social hierarchy. Although the prevalence of anger may be reduced from historical highs, it remains common in work and family situations (Chapter 19 by Scheiman, this book). Recent research addressing anger as a function of relative social status (e.g., Stets & Tushima, 2001) indicates that angry people will approach and confront subordinates, but retreat from and avoid superiors (Fitness, 2000; Kuppens, Van Mechelen, & Meulders, 2004). The observation that anger is more frequently and unilaterally, or at least non-reciprocally, expressed by members of an overclass against members of an underclass is not new and its effects go beyond just modes of expression. In the *Illiad*,

“A prince is stronger when he *choesthai* with (expresses anger at) an inferior man. . .” (Cairns, 2003). This opinion was shared by medieval European commentators on the power of kings (Hyams, 1998); lesser lords, too, ruled by judicious expressions of anger (Barton, 1998). Conversely, Aquinas, like Aristotle, observed that impositions by those with much greater status and power do not routinely engender anger in the imposed upon. More recent sociologically informed analyses are replete with references to the anger of the lower classes, how it fuels demonstrations and revolts, and how it must be harnessed for change. Accordingly, as Harris (2001) notes, advice to lower classes to restrain anger serves the interest of the upper class. Stearns and Stearns (1986) propose that, from one perspective, this amounts to a class-based solution to the social problem of anger control that works by obedience to authority rather than by the self-control practiced in some of the various band and village cultures noted above. They note that this process begins with parental “will-breaking” of children that then generalizes to obedience to social authorities. The frequent expression of anger by members of the empowered elite, and the seemingly natural (but perhaps developmentally shaped) suppression of anger and resignation toward those of higher status, may systematically function to maintain the status quo of class-based dominance and control. The ancient writers may not have had the framework of structural sociology in which to view instances of anger toward slaves, but they clearly had class-based interpretations of anger.

2.4 Summary

The enormous impact that anger has had on people and their social interactions has been recorded in many ways and in many places throughout history. As we review the impressive gains in scientific knowledge about anger in fields from anthropology to neuroscience, we recognize in contemporary hypotheses and experimental data the historical insights generated by the long line of scholars of which we are a part. This book expresses the continuity of this community.

Note

1. Compendium of Muslim Texts, University of Southern California <http://www.usc.edu/dept/MSA>

References

- Alés, C. (2000). Anger as a marker of love: The ethic of conviviality among the Yanomami. In J. Overing & A. Passes (Eds.), *The anthropology of love and anger: The aesthetics of conviviality in native Amazonia* (pp. 133–151). London: Routledge.
- Allen, D. S. (2003). Angry bees, wasps and jurors: The symbolic politics of ὄργη in Athens. In S. Braund & G. W. Most (Eds.), *Ancient anger perspectives from Homer to Galen* (Volume XXXII, pp. 76–98). Cambridge: Cambridge University Press.
- Baker, K. (2000). The temper thing. *American Heritage*, 51, 86–89.
- Barlow, S. A. (1989). Stereotype and reversal in Euripides' *Medea*. *Greece & Rome*, 36, 158–171.
- Barton, R. E. (1998). Zealous anger and the renegotiation of aristocratic relationships in eleventh and twelfth century France. In B. H. Rosenwein (Ed.), *Anger's past: The social uses of an emotion in the middle ages* (pp. 153–170). Ithaca, NY: Cornell University Press.
- Beck, R., & Fernandez, E. (1998). Cognitive-behavioral therapy in the treatment of anger: A meta-analysis. *Cognitive Therapy and Research*, 22, 63–74.

- Belaunde, L. E. (2000). The convivial self and the fear of anger amongst the Airo-Pai of Amazonian Peru. In J. Overing & A. Passes (Eds.), *The anthropology of love and anger: The aesthetics of conviviality in native Amazonia* (pp. 209–220). London: Routledge.
- Bellers, J. (1702). *A caution against all perturbations of the mind; anger as an enemy of the soul*. London: ATO.
- Briggs, J. L. (1970). *Never in anger: Portrait of an Eskimo family*. Cambridge: Harvard University Press.
- Buchan, T. (1980). Stranger in a strange land. *Zambezia*, 8, 149–158.
- Cairns, D. L. (2003). Ethics, ethology, terminology: Illadic anger and the cross-cultural study of emotion. In S. Braund & G. W. Most (Eds.), *Ancient Anger* (Volume XXXII, pp. 11–49). Cambridge: Cambridge University Press.
- Carr, J. E., & Tan, E. K. (1976). In search of the true amok: Amok as viewed within the Malay culture. *American Journal of Psychiatry*, 133, 1295–1299.
- Cohen, D., Nisbett, R. E., Bowdle, B. F., & Schwarz, N. (1994). Insult, aggression, and the southern culture of honor: An “experimental ethnography”. *Journal of Personality and Social Psychology*, 70, 945–960.
- Considine, P. (1986) The etymology of $\mu\eta\upsilon\lambda\zeta$ In: Studies in Honour of T.B.L. Webster I. Bristol, pp. 53–64
- Davies, N. (1981). *Human sacrifice – in history and today*. New York: William Morrow & Co.
- Del Vecchio, T., & O’Leary, K. D. (2004). Effectiveness of anger treatments for specific anger problems: A meta-analytic review. *Clinical Psychology Review*, 24, 15–34.
- Diamond, S. A. (1996). *Anger, madness, and the daimonic*. Albany, NY: State University of New York Press.
- DiGuiseppe, R., & Tafrate, R. C. (2003). Anger treatments for adults: A meta-analytic review. *Clinical Psychology: Science and Practice*, 10, 70–84.
- Dolcos, F., & McCarthy, G. (2006). Brain systems mediating cognitive interference by emotional distraction. *Journal of Neuroscience*, 26, 2072–2079.
- Dunbar, R. I. M., Clark, A., & Hurst, N. L. (1995). Conflict and cooperation among the Vikings: Contingent behavioral decisions. *Ethology and Sociobiology*, 16, 233–246.
- Duverger, C. (1983). *La flor letal. Economía del sacrificio azteca*. Mexico: Fondo de Cultura Económica.
- Eckhardt, C. I., & Deffenbacher, J. L. (1995). Diagnosis of anger disorders. In H. Kassirnové (Ed.), *Anger Disorders: Definition, diagnosis and treatment* (pp. 27–48). Washington, DC: Taylor & Francis.
- Ellis, A., & Dryden, W. (1987). *The practice of rational-emotive therapy*. New York: Springer.
- Fabing, H. D. (1956). On going berserk: A neurochemical inquiry. *American Journal of Psychiatry*, 113, 409–415.
- Fava, M. (1998). Depression with anger attacks. *Journal of Clinical Psychiatry*, 59, (518), 18–22.
- Ferracuti, S., Sacco, R., & Lazzari, R. (1996). Dissociative trance disorder: clinical and Rorschach findings in ten persons reporting demon possession and treated by exorcism. *Journal of Personality Assessment*, 66, 525–540.
- Fitness, J. (2000). Anger in the workplace: An emotion script approach to anger episodes between workers and their superiors, co-workers and subordinates. *Journal of Organizational Behavior*, 21, 147–162.
- Freedman, P. (1998). Peasant anger in the late middle ages. In B. H. Rosenwein (Ed.), *Anger’s Past: The Social Uses of an Emotion in the Middle Ages* (pp. 171–190). Ithaca, NY: Cornell University Press.
- Freud, S. (1933). *New introductory lectures on psycho-analysis* (pp. 82, 95). New York: W.W. Norton & Co.
- Froud, B., & Lee, A. (1978). *Faeries*. New York: Peacock Press/Bantam, ISBN 0-553-01159-6.
- Galen, C. (1963). *On the passions and errors of the soul* (P. W. Harkins, Trans.). Columbus, OH: Ohio State University Press.
- Gaw, A. C., & Bernstein, R. L. (1992). Classification of amok in DSM-IV. *Hospital and Community Psychiatry*, 43, 789–793.
- Ghazzal, Z. (1998). From anger on behalf of God to “forbearance” in Islamic medieval literature. In B. H. Rosenwein (Ed.), *Anger’s past: The social uses of an emotion in the middle ages* (pp. 203–232). Ithaca NY: Cornell University Press.
- Goldwasser, O. (2005). Where is metaphor? Conceptual metaphor and alternative classification in the hieroglyphic script. *Metaphor and Symbol*, 20, 95–113.
- Groark, K. (2008). Social opacity and the dynamics of empathic in-sight among the Tzotzil Maya of Chiapas, Mexico. *Ethos*, 36(4), 36, 427–448.
- Hall, G. S. (1899). A study of anger. *American Journal of Psychology*, 10, 516–591.
- Hanh, T. N. (2001). *Anger: Wisdom for cooling the flames*. New York: Riverhead Books.
- Hanson, A. E. (2003). “Your mother nursed you on bile”: Anger in babies and small children. In S. Braund & G. W. Most (Eds.), *Ancient anger: Perspectives from Homer to Galen* (Volume XXXII, pp. 185–207). Cambridge: Cambridge University Press.
- Harkins, J., & Wierzbicka, A. (2001). *Emotions in crosslinguistic perspective*. In J. Harkins & A. Wierzbicka (Eds.), New York: Mouton de Gruyter.

- Harris, G. G. (1978). *Casting out anger: Religion among the Taita of Kenya*. Cambridge: Cambridge University Press.
- Harris, W. H. (2001). *Restraining rage: The ideology of anger control in classical antiquity*. Cambridge: Harvard University Press.
- Harrison, R. K. (1979). Anger. In W. B. Geoffrey (Ed.), *The international standard Bible encyclopedia* (Vol. 1, pp. 127). Grand Rapids, MI: Eerdmans.
- Hercus, C. (1999). Identity, emotion and feminist collective action. *Gender & Society*, 13, 34–55.
- Hollan, D. (1988). Staying “Cool” in Toraja: Informal strategies for the management of anger and hostility in a nonviolent society. *Ethos*, 16, 52–72.
- Homer . (2004). *The Iliad* (R. Fitzgerald, Trans.). New York: Farrar, Straus and Giroux.
- Hupka, R. B., Lenton, A. P., & Hutchison, K. A. (1999). Universal development of emotion categories in natural language. *Journal of Personality and Social Psychology*, 77, 247–278.
- Hyams, C. P. (1998). What did Henry III of England think in bed and in French about kingship and anger? In B. H. Rosenwein (Ed.), *Anger's past: The social uses of an emotion in the middle ages* (pp. 92–126). Ithaca, NY: Cornell University Press.
- Irwin, J. R. (1947). Galen on the temperaments. *Journal of General Psychology*, 36, 45–64.
- Isaacs, C. T. (1987). The possessive states disorder: The diagnosis of demonic possession. *Pastoral Psychology*, 35, 263–273.
- Kemp, S., & Strongman, K. T. (1995). Anger theory and management: A historical analysis. *American Journal of Psychology*, 108, 397–417.
- Kendall, L. (1985). *Shamans, housewives, and other restless spirits: Women in Korean ritual life*. Honolulu: University of Hawaii Press.
- Kidd, S. (2000). Knowledge and the practice of love and hate among the Enxet of Paraguay. In J. Overing & A. Passes (Eds.), *The anthropology of love and anger: The aesthetics of conviviality in native Amazonia* (pp. 114–132). London : Routledge.
- Kon, Y. (1994). Amok. *British Journal of Psychiatry*, 165, 685–689.
- Kuppens, P. I., Van Mechelen, I. V., & Meulders, M. (2004). Every cloud has a silver lining: Interpersonal and individual differences determinants of anger-related behaviors. *Personality & Social Psychology Bulletin*, 30, 1550–1564.
- Lai, W. (1990). Looking for Mr. Ho Po: Unmasking the river god of ancient China. *History of Religions*, 29, 335–350.
- Lansky, M. R. (1996). Shame and suicide in Sophocles' Ajax. *Psychoanalytic Quarterly*, LXV, 761–786.
- Leslie, C. M. (1998). *Asian medical systems: A comparative study*. Delhi: Motilal Banarsidass Publ.
- Levy, R. I. (1973). *Tahitians: Mind and experience in the society islands*. Chicago: University of Chicago Press.
- Loewenstein, G. (2000). Emotions in economic theory and economic behaviour. *The American Economic Review*, 90, 426–432.
- Lutz, C. (1988). *Unnatural emotions*. Chicago: University of Chicago Press.
- Maalej, Z. (2004). Figurative language in anger expressions in tunisian Arabic: An extended view of embodiment. *Metaphor and symbol*, 19, 51–75.
- Markus, H. R., & Kitayama, S. (1991). Culture and the self-implications for cognition, emotion, and motivation. *Psychological Review*, 98, 224–253.
- Marti, B. (1945). Seneca's tragedies: A new interpretation. *Transactions and Proceedings of the American Philological Association*, 76, 216–245.
- Mikalsen, J. D. (1986). Zeus the father and Heracles the son in tragedy. *Transactions of the American Philological Association*, 116, 89–98.
- Miller, J. (1975). Kwulakan: The Delaware side of their movement west. *Pennsylvania Archeologist*, 45, 45–46.
- Most, G. (2003). Anger and pity in Homer's Iliad. In S. Braund & G. W. Most (Eds.), *Ancient anger perspectives from Homer to Galen* (Volume XXXII, pp. 50–75). Cambridge: Cambridge University Press.
- Myers, F. R. (1988). The logic and meaning of anger among Pintupi Aborigines. *Man*, 23, 589–610, New Series.
- Ohlander, S. (1989). *Dramatic suspense in Euripides' and Seneca's Medea*. New York: Peter Lang.
- Overing, J., & Passes, A. (2000). *The anthropology of love and anger: The aesthetics of conviviality in native Amazonia*. London: Routledge.
- Paludan, P. S. (2006). Lincoln and Negro slavery: I haven't got time for the pain. *Journal of the Abraham Lincoln Association*, 27, <http://www.historycooperative.org/journals/jala/27.2/paludan.html>.
- Phillips, D. A., VanPool, C. S., & VanPool, T. L. (2006). The horned serpent tradition in the North American Southwest. In C. S. VanPool, T. L. Vanpool, & D. A. Phillips (Eds.), *Religion in the prehispanic Southwest* (pp. 17–30). Lanham, MD: AltaMira Press.
- Plato. (1975). *Phaedrus and letters VII and VIII* (p. 62). Harmondsworth: Penguin Classics.

- Reid, J. P. (2006). *St Thomas Aquinas Summa Theologiae: 1a2ae. 40–48 Vol. 21 Fear and Anger*. Cambridge: Cambridge University Press.
- Rosenwein, B. H. (1998). *Anger's past: The social uses of an emotion in the middle ages*. Ithaca, NY: Cornell University Press.
- Rydstrom, H. (2003). Encountering “hot” anger: Domestic violence in contemporary Vietnam. *Violence Against Women*, 9, 676–697.
- Santos-Graneros, F. (2000). The Sisyphus syndrome, or the struggle for conviviality in native Amazonia. In J. Overing & A. Passes (Eds.), *The anthropology of love and anger: The aesthetics of conviviality in Native Amazonia*. (pp. 268–287). London: Routledge.
- Seneca, L. (44/1817). *Seneca's morals*. New York: Harper & Brothers.
- Simon, B. (1978). *Mind and madness in ancient Greece: The classical roots of modern psychiatry*. Ithaca, NY: Cornell University Press.
- Singer, I. (2002). *Hittite prayers*. The Netherlands: Brill Leiden.
- Somer, E., & Saadon, M. (2000). Stombali: Dissociative possession and trance in a tunisian healing dance. *Transcultural Psychiatry*, 37, 581–602.
- Speidel, M. P. (2002). Berserks: A history of Indo-European “mad warriors”. *Journal of World History*, 13, 253–290.
- Spores, J. C. (1988). *Running amok: An historical inquiry*. Athens, OH: Ohio University, Monographs in International Studies, Southeast Asia Series, No. 82.
- Stearns, C. Z., & Stearns, P. N. (1986). *Anger: The struggle for emotional control in America's history*. Chicago: University of Chicago Press.
- Stets, J. E., & Tsushima, T. M. (2001). Negative emotion and coping responses within identity control theory. *Social Psychology Quarterly*, 64, 283–295.
- Sukhodolsky, D. G., Kassino, H., & Gorman, B. S. (2004). Cognitive-behavior therapy for anger in children and adolescents: A meta-analysis. *Aggression and Violent Behavior*, 9, 247–269.
- Tedlock, B. (1983). American Indian religions. *American Indian Quarterly*, 7, 93–110.
- Tierney, P. (1989). *The highest altar: The story of human sacrifice*. New York: Viking.
- Trask, R. (1975). *Salem village and the witch hysteria*. New York: Grossman.
- Tsytarev, S. V., & Grodnitzky, G. R. (1995). Anger and criminality. In H. Kassino (Ed.), *Anger disorders: Definition, diagnosis, and treatment* (pp. 91–108). Boca Rotan, FL: Taylor & Francis.
- Verneze, P. J. (2008). Moderation or the middle way: Two approaches to anger. *Philosophy East & West*, 58, 2–16.
- Wescott, J., & Morton-Williams, P. (1962). The symbolism and ritual context of the Yoruba Laba Shango. *The Journal of the Royal Anthropological Institute of Great Britain and Ireland*, 92, 23–37.

Part II

Biology of Anger

Chapter 3

Population and Molecular Genetics of Anger and Aggression: Current State of the Art

Martin Reuter

Abstract Twin and adoption studies have demonstrated that about half of the variance in the aggression–hostility–anger (AHA) syndrome can be accounted for by genes. This strong heritability gives rise to the question which genes influence this facet of our personality that determines the proclivity for being aggressive or angry. Due to revolutionary progress in the field of molecular genetics over the last years first candidate genes for AHA have been identified. This chapter gives a short introduction in population and molecular genetics of anger and aggression. The most influential findings are reviewed. It becomes apparent that besides variations on genes coding for gonadal hormones, polymorphisms of serotonergic and dopaminergic neurotransmitter genes seem to be more relevant. Future perspectives in molecular genetic research are discussed that will help to unravel the genetic underpinnings of anger and aggression.

3.1 Preface

The present book is an interdisciplinary overview of the essential scientific findings related to anger. Molecular genetic research is the youngest discipline that addresses the foundations of basic emotional systems. The endeavor to identify the molecular basis of anger has just begun and it has been the most often studied in the context of aggression. Therefore, this chapter will treat anger in that context. Anger and aggression overlap to a great extent, although one can be aggressive without being angry and one can be angry without being aggressive. Psychophysiological studies have a much longer tradition and here the discrimination between anger and aggression by parameters of the autonomic nervous system is at least partly successful (Chapter 7 by Stemmler, this book). Molecular genetic research with respect to anger and aggression originates from the subfield of psychiatry in which the main interest is in psychopathologies associated with overt aggression. Anger in the form of inwardly directed negative emotionality has been only of secondary interest in this research domain. Moreover, phenotypes investigated in this field (e.g., the Buss–Durkee Hostility Inventory, BDHI; one of the most popular aggression/anger scales; Buss & Durkee, 1957) that were associated with certain gene loci or gene regions are broadly defined encompassing mostly a mixture between anger and aggression. These studies generally focus on hostility and angry

M. Reuter (✉)
Department of Psychology, University of Bonn, Bonn, Germany
e-mail: martin.reuter@uni-bonn-diff.de

aggression (as opposed to instrumental/proactive aggression such as bullying). This frequently co-occurring mix of aggression, hostility, and anger has been termed the AHA syndrome (Chapter 23 by Spielberg & Reheiser, this book). Because the literature on AHA is more extensive (and the differentiation between aggression and anger has often been ignored), this chapter will deal with the molecular genetic basis of aggression and AHA.

3.2 Introduction

Gender differences with respect to aggressive behaviors are the most salient indicators for the involvement of genetic factors in the expression of aggression (Gatewood et al., 2006). Males have significantly higher scores in aggression and show more deviant behaviors. This finding of higher aggression in males is consistent across different animal species and is also valid for humans, at least for physical aggression. Sexual dimorphisms in a certain phenotype implicate the Y-chromosome that determines gender and is primarily responsible for the masculinization of an individual. Here, the gonadal steroid hormone testosterone plays a crucial role. It is synthesized in the testis in males and in the adrenals in both sexes. Blood concentrations of testosterone are ten times higher in human males than in females and are assumed to determine most of the variance in sex differences with respect to aggression. Animal studies have shown that testosterone levels also determine the likelihood of aggression in males. Testosterone levels have been related to social rank, physical strength, and extent of aggressive behaviors (Sapolsky, 1991). Conversely, animals that become subordinate in struggles for sexual partners or positions in social hierarchies react with a decrease in testosterone levels after defeat (e.g., Setchell & Dixon, 2001). If testosterone is one of the most important facilitators of aggressive behaviors, the decline in its levels across the life span should be accompanied by a corresponding decline in aggression. In fact, aggression does decline significantly with age. This is also the case for other personality variables like extraversion and neuroticism that are presumably determined primarily by neurotransmitter activity instead of testosterone (McCrae et al., 1999). Therefore, it appears that a reduction in the metabolism of biological systems is congruent with a reduced expression of associated personality traits. Variability of biological systems is strongly determined by genetic factors that regulate anabolic and catabolic enzyme activity, reuptake into presynaptic nerve vesicles, and receptor density and sensitivity.

3.3 The Heritability of Aggression and Hostility

The heritability of a given phenotype can be estimated from adoption and twin studies. The multivariate statistical designs for such population genetics studies do not depend on any molecular genetic data. In twin studies, one method of estimating heritability is to compare correlation coefficients between mono- and dizygotic twins. Due to the fact that monozygotic twins share 100% of the same genome and dizygotic twins have only 50% of the genetic material in common, a simple formula which estimates heritability (h^2) of a given phenotype is twice the difference of the intra-pair correlation coefficients of mono- and dizygotic twins ($h^2 = 2(r_{MZ} - r_{DZ})$). Adoption studies compare the resemblance between adopted children and their biological parents/sibs with the resemblance between adopted children and their stepparents/stepparents. The total variance in a given phenotype is then separated into genetic influences and influences of shared and non-shared environments, plus a measurement error representing a lack of reliability of the method by which the phenotype is assessed.

Twin and adoption studies have proven that personality traits are strongly heritable (about 50% of the variance is explained by genes) but that the influence of environmental factors is equally strong (e.g., Bouchard, 1994). This simple division into heredity and environment is complicated by a number of factors. Chief among these are that individuals not only share 50% of their genome with their parents, but they are also influenced by an environment that is shaped by the same parents who also provide the genes. This effect is called gene–environment interaction. The influence of the shared environment is especially strong during childhood, but becomes weaker with age as individuals build their own environment under the influence of genetic factors. There are now a number of quantitative genetic studies that have assessed the heritability of aggression, anger, and irritability. In a twin study on 182 monozygotic and 118 dizygotic Vietnamese twins, Coccaro, Bergeman, Kavoussi, and Seroczynski (1997) reported that up to 47% in the variance in the subscales of the Buss–Durkee Hostility Inventory were accounted for by genetic influences. Because some of these BDHI scales have been shown to correlate with indices of central 5-HT function, the authors conclude that impulsive aggression, as reflected by these scales, is heritable in men. In a second twin study comparing monozygotic and dizygotic twins reared together and apart, Coccaro, Bergeman, and McClearn (1993) demonstrated a substantial genetic influence on two factors of aggression, lack of assertiveness/aggression and impulsive irritability, whereas there was no effect for shared-environmental influences for both factors.

3.4 Molecular Genetic Research in Psychology

What are the genetic mechanisms that give rise to the heritability of AHA? The invention of the polymerase chain reaction (PCR) was the first of many new techniques in molecular genetics that have permitted the investigation of the genetic basis of psychological phenotypes. The flood of findings increases in parallel to the technical progress in this area. Gene-oriented research profits from the high reliability of genetic techniques. Once a protocol for the genotyping of a certain polymorphism (a variation at a certain gene locus that is manifested by the occurrence of different alleles) has been established, the reliability is perfect. The reliability of psychological measurements like questionnaires is far from perfect. Internal consistencies of .70 are considered satisfactory, but this still leaves large and problematic unexplained variance and error variance. It is this error variance that complicates the identification of candidate genes for a certain phenotype.

In genetic association studies the genotype or allele frequencies in a certain gene locus are related to the scores in a certain quantitative trait (e.g., an aggression scale) or a certain categorical phenotype (incarcerated due to assault vs. having no conflict with the law). Although the reliability of the predictor (genotype or allele frequencies) is not contaminated by error variance, there are many inconsistent findings in genetic association studies. The major problems here are stratification errors (the drawing of an unrepresentative sample) or inhomogeneity with respect to ethnicity. Allele frequencies can vary substantially across different ethnic samples and therefore each ethnicity mixture in a sample can influence the outcome of the study. Multi-ethnic countries, e.g., the USA, must strictly control for ethnicity.

3.5 Variations in Genes Coding for Gonadal Hormones

Besides differences in sex chromosomes, allelic variations on genes coding for gonadal hormones have also been investigated with respect to aggressive behaviors. In mice positive associations between the estrogen receptor alpha gene (Trainor, Greiwe, & Nelson, 2006), the estrogen

receptor beta gene (Nomura et al., 2006), and the androgen receptor gene (for a review see Robins, 2005) and aggression have been found. With respect to humans Westberg et al. (2003) reported an association between a dinucleotide repeat polymorphism of the estrogen receptor alpha gene and non-conformity, irritability, suspicion, and psychoticism in a sample of healthy female subjects. Jonsson et al. (2001) found a trend for an association between an androgen receptor trinucleotide repeat polymorphism and personality traits related to dominance and aggression. In a replication study cross-validating associations between the number of CAG repeats (repetition of a particular nucleotide triplet) in the androgen receptor gene and psychological traits related to aggression in a Swedish and an Australian sample, Loehlin, Jonsson, Gustavsson, Schalling, and Stallings (2003) concluded that if the number of CAG repeats on this gene were related to psychological traits at all, the relationship is a weak one.

3.6 The Influence of Serotonergic Gene Loci on Aggression and Anger

The fact that testosterone cannot account for the total variance in AHA implies that other biological factors must be involved. A seminal study by Asberg and Traskman (1981) that detected an association between low central nervous serotonin (5-HT) levels and AHA provided first evidence for the importance of the 5-HT system in aggression.

Several other pharmacological and endocrinological studies supported this hypothesis. Especially variance within gender groups could be explained by the neurotransmitter activity of the 5-HT system. Brown, Goodwin, Ballenger, Goyer, and Major (1979) reported an association between the level of aggression in males and 5-HIAA (the major metabolite of 5-HT) levels in the cerebrospinal fluid. Bioulac, Benezech, Renaud, Roche, and Noel (1978) found a significant decrease in the 5-HIAA turnover in patients with the XYY syndrome who represent a high percentage of patients admitted to forensic facilities for aggressiveness. This finding provides evidence for a relationship between the Y-chromosome and the 5-HT system. Based on these and other studies the search for the molecular genetic basis of aggressive behaviors concentrated on genes related to the 5-HT system. One of the most prominent candidate genes for aggression so far is the tryptophan hydroxylase (TPH) 1 gene. TPH is the rate limiting biosynthetic enzyme in the serotonin pathway and regulates levels of 5-HT by converting tryptophan into 5-hydroxytryptophan, which is the direct precursor of 5-HT.

Two polymorphisms on intron 7 of the TPH 1 gene, A218C and A779C have been related to aggression and suicide across different studies. Suicide is considered as an extreme form of auto-aggression. However, this review will just focus on outwardly directed forms of anger and aggression.

Table 3.1 gives a chronologically ordered overview of genetic association studies relating to the TPH1 gene to aggression-related behaviors. Two polymorphisms, A779C and A218C, have been shown to be in strong linkage disequilibrium (Nielsen et al., 1998). That is, a certain haplotype of the A218C and the A779C is found in nearly 100% of the population, indicating a deviation from the genetic processes of random recombination. Therefore, one cannot expect different associations between the one and the other with behavior or other outcome variables.

Several studies found associations between the A779C polymorphism and lowered cerebrospinal fluid (CSF) 5-HIAA levels. Lower CSF 5-HIAA levels in healthy men, but not in healthy women, carrying the TPH A have been reported (Jönsson et al., 1997). The lowest CSF 5-HIAA levels were found in a sample of impulsive alcoholic violent offenders with the CC-genotype (Nielsen et al., 1994). Such inconsistent results occur not only in these functional studies, which relate the TPH1 polymorphisms to 5-HT activity, but also in association studies relating the TPH1 single nucleotide polymorphisms (SNPs) to measures of AHA (see Table 3.1). However, a trend for the impact of

Table 3.1 Associations between SNPs of the TPH1 gene (A218C, A779C) and aggression-related behaviors and indicators of 5-HT activity

Authors	Sample	Phenotype	Results
Reuter and Hennig (2005a)	Healthy subjects (<i>n</i> = 252)	Buss–Durkee–Hostility Inventory (BDHI)	Positive heterosis effect for the A779C SNP
Hennig, Reuter, Netter, Burk, and Landt (2005)	Healthy non-smokers (<i>n</i> = 58)	Buss–Durkee–Hostility Inventory (BDHI)	Association between the AA-genotype of the A779C SNP and aggressive hostility but not neurotic hostility
Tsai et al. (2003)	<i>n</i> = 209 (103 men and 106 women) healthy subjects	TPQ personality scales	No associations
Rujescu et al. (2002)	Healthy volunteers (<i>n</i> = 154) and suicide attempters (<i>n</i> = 86)	State anger, trait anger, and angry temperament	Association with the A-allele in both samples
Staner et al. (2002)	Inpatients with impulsive behavioral tendencies (<i>n</i> = 54) vs. nonimpulsive controls (<i>n</i> = 27)	Impulsive aggressive behavior	Association with the 218C allele
Nolan, Volavka, Lachman, and Saito (2000)	Men and women with schizophrenia or schizoaffective disorder (<i>n</i> = 84)	History of either assaultive or threatening behavior	Association with the C-allele in males
Manuck et al. (1999)	Healthy subjects (<i>n</i> = 251)	Aggression and anger-related traits of personality	Association with the A-allele (A218C)
Nielsen et al. (1998)	<i>n</i> = 804 impulsive alcoholic offenders, controls, and their relatives (369 sib pairs)	Alcoholism, suicidality, personality socialization, 5-HIAA levels	C-allele is associated with suicidality, alcoholism, and Karolinska Scales of Personality Socialization, no association with 5-HIAA levels
New et al. (1998)	<i>n</i> = 21 males with personality disorder	BDHI personality scores	Highest assault and irritability scores in CC-carriers
Jönsson et al. (1997)	<i>n</i> = 66 healthy volunteers (men and women)	5-HIAA levels	Lowered 5-HIAA levels in men but not in women carrying the A-allele
Mann et al. (1997)	<i>n</i> = 51 patients with major depression, with or without a history of suicidal acts	Suicide attempts, 5-HIAA levels	A-allele occurred with greater frequency in the patients who had attempted suicide; no association with 5-HIAA levels
Nielsen et al. (1994)	<i>n</i> = 56 impulsive and <i>n</i> = 14 nonimpulsive alcoholic violent offenders, <i>n</i> = 20 healthy volunteers	5-HIAA levels	In the impulsive alcoholic violent offenders group: lowest 5-HIAA levels in CC-carriers

the A-allele on AHA is apparent and was supported by a meta-analysis (Rujescu, Giegling, Sato, Hartmann, & Moller, 2003).

Other serotonergic gene markers have also been investigated with respect to AHA. Using a case–control design, Sakai et al. (2006) found an association between the ss-genotype of the serotonin transporter polymorphism (5-HTTLPR) and conduct disorder. 5-HTTLPR is an insertion deletion polymorphism in the promoter region of the serotonin transporter gene. Carriers of the short s-allele lack a sequence of 43-base pairs. The s-allele is related to reduced transcription rate, 5-HT transporter density, and 5-HT reuptake (Lesch et al., 1996; Greenberg et al., 1999).

Using a family design separate from their case–control design, Sakai et al. (2006) also found an association between the s-allele and aggressive symptoms. In line with these findings, Gerra et al. (2005) reported an association between the ss-genotype of the 5-HTTLPR and aggressive novelty seeking and total BDHI score. These findings were supported by a study on clinically referred children displaying extreme aggression (Beitchman et al., 2006) as well as by a family-based association test in a general population study of AHA (Haberstick, Smolen, & Hewitt, 2006). Imaging studies using positron emission tomography (PET) corroborated the molecular genetic findings indicating an association between AHA and the s-allele of the 5-HTTLPR. Frankle et al. (2005) reported a lower brain serotonin transporter density and binding potential – especially in the anterior cingulate cortex (ACC) – in subjects with impulsive aggressivity.

Zuckerman (1984) proposed a personality trait of Psychoticism Unsocialized Sensation Seeking (PIMPUSS) that is related to impulsivity, the seeking of new sensations, risk-taking behavior, boredom susceptibility, aggressiveness, and unsocialized behavior. Not surprisingly PIMPUSS shows a marked gender dimorphism with higher scores in men than in women. As expected, PIMPUSS was related to higher testosterone levels in males. Other biological markers of sensation seeking are lowered norepinephrine levels (NE) and monoamine oxidase-A (MAO-A) levels. MAO is an enzyme that degrades NE and 5-HT.

Based on these findings, variants on the MAO-A gene are candidate loci for aggression. Interestingly, the MAO-A gene is located on the X-chromosome (Xp11.5), which implies gender differences in phenotypes related to MAO activity. Manuck, Flory, Ferrell, Mann, and Muldoon (2000) reported an association between a regulatory polymorphism of the MAO-A gene and aggression and impulsivity. Participants with two or three repeats in this “variable number of tandem repeats” (VNTR) polymorphism had not only higher aggression and impulsivity scores but also a blunted 5-HT response (as measured by prolactin) to a pharmacological challenge test with fenfluramine hydrochloride (a 5-HT releaser/reuptake inhibitor). In line with this finding, Gerra et al. (2004) reported a higher frequency of the 3-allele in the MAO-A promoter VNTR polymorphism in violent offenders among heroin addicts, compared to addicted individuals without antisocial behavior. Moreover, the 3-repeat-allele was associated with higher BDHI scores. It could be demonstrated that alleles with 3.5 and 4 repeats are 2–10 times more efficient in metabolizing monoamines than the 3-repeat allele with respect to MAO activity (Contini, Marques, Garcia, Hutz, & Bau, 2006). In a sample of 125 Brazilian alcoholics of European descent and 235 controls the 3-repeat allele was associated with alcohol dependence, comorbid drug abuse among alcoholics, and a higher number of antisocial symptoms. However, Widom and Brzustowicz (2006) found no main effect for the MAO-A gene with respect to violent and antisocial behavior. In a functional study Ducci et al. (2006) found an association between DA metabolism and MAO-VNTR allelic variation. Alleles conferring high activity were related to higher HVA (homovanillic acid, a major metabolite of catecholamine metabolism) levels in CSF; in contrast no associations with the respective NE and 5-HT metabolites MHPH or 5-HIAA were detected.

The cited studies were supported by animal research using knockout mice as a powerful research tool to prove the association between MAO-A activity and aggression. Mice with an MAO-A gene knockout showed increased aggression. However, knockout studies also demonstrate the complexity of neurochemical regulation. A lack of the MAO-A gene is accompanied by downregulation of the serotonergic receptors 5-HT1A, 5-HT2A, and 5-HT2C (for a review see Shih, 2004); this downregulation presumably reflects compensatory, homeostatic regulation of 5-HT activity.

3.7 Catecholamine-Associated Gene Loci and AHA

Whereas MAO degrades monoamines (5-HT, DA, NE) in presynaptic nerve terminals, catechol-*O*-methyltransferase (COMT) is involved in the catabolism of catecholamines (DA, NE) in the synaptic cleft. Most of the available evidence indicates that norepinephrine and dopamine lower the threshold for an aggressive response to environmental stimuli. It was hypothesized that if AHA-related behavior is enhanced by catecholaminergic activity, then the lower activity of COMT and MAO-A (resulting in a slower inactivation of catecholamines) should indirectly enhance aggression (Volavka, Bilder, & Nolan, 2004). With respect to COMT a single nucleotide polymorphism (SNP), a G→A transition in codon 158 of the COMT gene located at the q11 band of human chromosome 22, results in three- to fourfold difference in COMT enzyme activity by coding for the synthesis of the amino acid methionine (MET) instead of valine (VAL). Carriers of the VAL/VAL genotype show highest enzyme activity, MET/MET carriers lowest, and heterozygotes (VAL/MET genotype) have intermediate levels of COMT activity (Lachman et al., 1996). Given the enormous functional effects resulting from the exchange of a single base, this gene locus has been associated with numerous phenotypes including cognitive functions, personality traits, and psychiatric disorders.

With respect to AHA-related personality traits there are many studies that have investigated the role of the COMT VAL158MET SNP in schizophrenic patients. Most of these studies found an association between the low activity MET-allele or the MET/MET genotype and AHA (Strous, Bark, Parsia, Volavka, & Lachman, 1997; Strous et al., 2003; Han, Park, Na, Kee, & Lee, 2004; Han et al., 2006; Lachman, Nolan, Mohr, Saito, & Volavka, 1998). Jones et al. (2001) are the only ones to report higher AHA scores in carriers of the VAL/VAL genotype. However, AHA was assessed only after the onset of schizophrenia so that it remains unclear if the gene–behavior association was operational during pre-morbid functioning or occurred only in the presence of the disease. In any case, these findings cannot be necessarily extrapolated to healthy subjects.

In a sample of healthy subjects Rujescu, Giegling, Gietl, Hartmann, and Moller (2003) observed that subjects with the MET/MET genotype showed more outwardly directed anger whereas the VAL/VAL genotype was related to more inwardly directed anger. In line with the results of Hennig et al. (2005; see Table 3.1) these findings suggest that anger is not a homogenous phenotype, but that subtypes must be considered that can be related to different alleles/genotypes at the same gene locus. Interestingly, the strongest association between the COMT VAL158MET SNP and personality was reported not with respect to AHA but with extraversion. Subjects homozygous for the VAL-allele had significantly higher extraversion scores than carriers of the MET allele (Reuter & Hennig, 2005b).

Comings et al. (2000) investigated the role of the noradrenergic system for AHA in an association study including a student sample and parents of twins in the Minnesota Twin Study. In a group of students a SNP in the promoter region of the alpha 2a (ADRA2a) receptor gene was associated with higher BDHI scores whereas in the parent group the same association could only be confirmed for measures of impulsivity that are related at least to a facet of AHA.

3.8 Summary and Future Perspectives

This chapter has presented an overview of the contributions made by genetic research to identify the biological basis of AHA. Starting from milestones in quantitative genetic research that clearly demonstrated that personality variables related to AHA are strongly affected by genetic factors (e.g., Bouchard, 1994), molecular genetic research has tried to identify gene loci that contribute

to this heritability. Chromosomal differences with respect to gonosomes begin to explain, at least roughly, the sexual dimorphism of AHA in which men show more overt and violent forms of behavior. Given that personality traits are determined by many genes, the identification of all relevant gene loci is difficult. This difficulty favors a candidate gene approach that focuses on single functional loci on genes that regulate neuroendocrine function and that have been related to the phenotypes under investigation. Differences in aggression within each gender have been related to variations in the availability of androgens and estrogens. Therefore, polymorphisms on genes coding for receptors of testosterone or estrogen have been investigated with respect to aggression. An association between dinucleotide repeat polymorphism of the estrogen receptor alpha gene and AHA-related traits was successfully demonstrated by Westberg et al. (2003). However, replication of this finding is still lacking. This is an important caveat, given that initial positive results associating an androgen receptor polymorphism with aggression have not been supported.

Stimulated by biochemical studies relating the 5-HT system to AHA, candidate genes of the 5-HT system have been extensively investigated. The TPH1 gene and the 5-HT transporter gene have consistently turned out to be associated with AHA. Furthermore, genes related to the metabolism of catecholamines, especially the MAO-A gene and the COMT gene, have also been linked to AHA.

Inconsistent results often stem from methodological shortcomings. Only representative samples with extremely large sample sizes controlling for differences in ethnicity can improve the quality of genetic association studies. A multi-method approach, combining genetic with functional data, is required for deeper insights into the biological underpinnings of AHA. Most of the association studies available depend on a limited number of gene loci that can only account for small proportions of variance in the phenotype. However, the future lies in genome-wide scans that allow associating several hundred thousand SNPs at a time with a given phenotype. Microarray techniques have already made this possible. Presently, the costs of such microchip-based genetic research are exorbitantly high and they are therefore applied mostly to phenotypes that are of direct clinical relevance (e.g., ADHD, schizophrenia; Hebebrand et al., 2006; Bulayeva, Glatt, Bulayev, Pavlova, & Tsuang, 2007) or medical diseases (e.g., diabetes, asthma, cancer; Smyth et al., 2006; Pillai et al., 2006; Bergman et al., 2007). However, it is only a question of time until these techniques are applied in personality research.

References

- Asberg, M., & Traskman, L. (1981). Studies of CSF 5-HIAA in depression and suicidal behaviour. *Advances in Experimental Medicine and Biology*, 133, 739–752.
- Beitchman, J. H., Baldassarra, L., Mik, H., De Luca, V., King, N., Bender, D., et al. (2006). Serotonin transporter polymorphisms and persistent, pervasive childhood aggression. *American Journal of Psychiatry*, 163, 1103–1105.
- Bergman, A., Karlsson, P., Berggren, J., Martinsson, T., Bjorck, K., Nilsson, S., et al. (2007). Genome-wide linkage scan for breast cancer susceptibility loci in Swedish hereditary non-BRCA1/2 families: Suggestive linkage to 10q23.32-q25.3. *Genes Chromosomes and Cancer*, 46, 302–309.
- Bioulac, B., Benezech, M., Renaud, B., Roche, D., & Noel, B. (1978). Biogenic amines in 47, XYY syndrome. *Neuropsychobiology*, 4, 366–370.
- Bouchard, T. J., Jr. (1994). Genes, environment, and personality. *Science*, 264, 1700–1701.
- Brown, G. L., Goodwin, F. K., Ballenger, J. C., Goyer, P. F., & Major, L. F. (1979). Aggression in humans correlates with cerebrospinal fluid amine metabolites. *Psychiatry Research*, 1, 131–139.
- Bulayeva, K. B., Glatt, S. J., Bulayev, O. A., Pavlova, T. A., & Tsuang, M. T. (2007). Genome-wide linkage scan of schizophrenia: A cross-isolate study. *Genomics*, 89, 167–177.
- Buss, A. H., & Durkee, A. (1957). An inventory for assessing different kinds of hostility. *Journal of Consulting Psychology*, 21, 343–349.
- Coccaro, E. F., Bergeman, C. S., Kavoussi, R. J., & Seroczynski, A. D. (1997). Heritability of aggression and irritability: A twin study of the Buss–Durkee aggression scales in adult male subjects. *Biological Psychiatry*, 41, 273–284.

- Coccaro, E. F., Bergeman, C. S., & McClearn, G. E. (1993). Heritability of irritable impulsiveness: A study of twins reared together and apart. *Psychiatry Research*, 48, 229–242.
- Comings, D. E., Johnson, J. P., Gonzalez, N. S., Huss, M., Saucier, G., McGue, M., et al. (2000). Association between the adrenergic alpha 2A receptor gene (ADRA2A) and measures of irritability, hostility, impulsivity and memory in normal subjects. *Psychiatric Genetics*, 10, 39–42.
- Contini, V., Marques, F. Z., Garcia, C. E., Hutz, M. H., & Bau, C. H. (2006). MAOA-uVNTR polymorphism in a Brazilian sample: Further support for the association with impulsive behaviors and alcohol dependence. *American Journal of Medical Genetics B Neuropsychiatric Genetics*, 141, 305–308.
- Ducci, F., Newman, T. K., Funt, S., Brown, G. L., Virkkunen, M., & Goldman, D. (2006). A functional polymorphism in the MAOA gene promoter (MAOA-LPR) predicts central dopamine function and body mass index. *Molecular Psychiatry*, 11, 858–866.
- Frankle, W. G., Lombardo, I., New, A. S., Goodman, M., Talbot, P. S., Huang, Y., et al. (2005). Brain serotonin transporter distribution in subjects with impulsive aggressivity: A positron emission study with [¹¹C]McN 5652. *American Journal of Psychiatry*, 162, 915–923.
- Gatewood, J. D., Wills, A., Shetty, S., Xu, J., Arnold, A. P., Burgoyne, P. S., et al. (2006). Sex chromosome complement and gonadal sex influence aggressive and parental behaviors in mice. *Journal of Neuroscience*, 26, 2335–2342.
- Gerra, G., Garofano, L., Bosari, S., Pellegrini, C., Zaimovic, A., Moi, G., & et al. (2004). Analysis of monoamine oxidase A (MAO-A) promoter polymorphism in male heroin-dependent subjects: behavioural and personality correlates. *Journal of Neural Transmission*, 111, 611–621.
- Gerra, G., Garofano, L., Castaldini, L., Rovetto, F., Zaimovic, A., Moi, G., et al. (2005). Serotonin transporter promoter polymorphism genotype is associated with temperament, personality traits and illegal drugs use among adolescents. *Journal of Neural Transmission*, 112, 1397–1410.
- Greenberg, B. D., Tolliver, T. J., Huang, S. J., Li, Q., Bengel, D., & Murphy, D. L. (1999). Genetic variation in the serotonin transporter promoter region affects serotonin uptake in human blood platelets. *American Journal of Medical Genetics*, 88, 83–87.
- Haberstick, B. C., Smolen, A., & Hewitt, J. K. (2006). Family-based association test of the 5HTTLPR and aggressive behavior in a general population sample of children. *Biological Psychiatry*, 59, 836–843.
- Han, D. H., Kee, B. S., Min, K. J., Lee, Y. S., Na, C., Park, D. B., et al. (2006). Effects of catechol-O-methyltransferase Val158Met polymorphism on the cognitive stability and aggression in the first-onset schizophrenic patients. *NeuroReport*, 17, 95–99.
- Han, D. H., Park, D. B., Na, C., Kee, B. S., & Lee, Y. S. (2004). Association of aggressive behavior in Korean male schizophrenic patients with polymorphisms in the serotonin transporter promoter and catecholamine-O-methyltransferase genes. *Psychiatry Research*, 129, 29–37.
- Hebebrand, J., Dempfle, A., Saar, K., Thiele, H., Herpertz-Dahlmann, B., Linder, M., et al. (2006). A genome-wide scan for attention-deficit/hyperactivity disorder in 155 German sib-pairs. *Molecular Psychiatry*, 11, 196–205.
- Hennig, J., Reuter, M., Netter, P., Burk, C., & Landt, O. (2005). Two types of aggression are differentially related to serotonergic activity and the A779C TPH polymorphism. *Behavioral Neuroscience*, 119, 16–25.
- Jones, G., Zammit, S., Norton, N., Hamshere, M. L., Jones, S. J., Milham, C., et al. (2001). Aggressive behaviour in patients with schizophrenia is associated with catechol-O-methyltransferase genotype. *British Journal of Psychiatry*, 179, 351–355.
- Jönsson, E. G., Goldman, D., Spurlock, G., Gustavsson, J. P., Nielsen, D. A., Linnoila, M., Owen, M. J., & Sedvall, G. C. (1997). Tryptophan hydroxylase and catechol-O-methyltransferase gene polymorphisms: relationships to monoamine metabolite concentrations in CSF of healthy volunteers. *European Archives of Psychiatry and Clinical Neuroscience*, 247, 297–302.
- Jonsson, E. G., von Gertten, C., Gustavsson, J. P., Yuan, Q. P., Lindblad-Toh, K., Forslund, K., et al. (2001). Androgen receptor trinucleotide repeat polymorphism and personality traits. *Psychiatric Genetics*, 11, 19–23.
- Lachman, H. M., Nolan, K. A., Mohr, P., Saito, T., & Volavka, J. (1998). Association between catechol O-methyltransferase genotype and violence in schizophrenia and schizoaffective disorder. *American Journal of Psychiatry*, 155, 835–837.
- Lachman, H. M., Papolos, D. F., Saito, T., Yu, Y. M., Szumlanski, C. L., & Weinshilboum, R. M. (1996). Human catechol-O-methyltransferase pharmacogenetics: Description of a functional polymorphism and its potential application to neuropsychiatric disorders. *Pharmacogenetics*, 6, 243–250.
- Lesch, K. P., Bengel, D., Heils, A., Sabol, S. Z., Greenberg, B. D., & Petri, S., (1996). Association of anxiety-related traits with a polymorphism in the serotonin transporter gene regulatory region. *Science*, 274, 1527–1531.
- Loehlin, J. C., Jonsson, E. G., Gustavsson, J. P., Schalling, M., & Stallings, M. C. (2003). The androgen receptor gene and psychological traits: Are results consistent in Sweden and Australia? *Twin Research*, 6, 201–208.

- Mann, J. J., Malone, K. M., Nielsen, D. A., Goldman, D., Erdos, J., & Gelernter, J. (1997). Possible association of a polymorphism of the tryptophan hydroxylase gene with suicidal behavior in depressed patients. *American Journal of Psychiatry*, *154*, 1451–1453.
- Manuck, S. B., Flory, J. D., Ferrell, R. E., Dent, K. M., Mann, J. J., & Muldoon, M. F. (1999). Aggression and anger-related traits associated with a polymorphism of the tryptophan hydroxylase gene. *Biological Psychiatry*, *45*, 603–614.
- Manuck, S. B., Flory, J. D., Ferrell, R. E., Mann, J. J., & Muldoon, M. F. (2000). A regulatory polymorphism of the monoamine oxidase-A gene may be associated with variability in aggression, impulsivity, and central nervous system serotonergic responsivity. *Psychiatry Research*, *95*, 9–23.
- McCrae, R. R., Costa, P. T., Jr., Pedrosa de Lima, M., Simoes, A., Ostendorf, F., Angleitner, A., et al. (1999). Age differences in personality across the adult life span: Parallels in five cultures. *Developmental Psychology*, *35*, 466–477.
- New, A. S., Gelernter, J., Yovell, Y., Trestman, R. L., Nielsen, D. A., Silverman, J., et al. (1998). Tryptophan hydroxylase genotype is associated with impulsive-aggression measures: A preliminary study. *American Journal of Medical Genetics*, *81*, 13–17.
- Nielsen, D. A., Goldman, D., Virkkunen, M., Tokola, R., Rawlings, R., & Linnoila, M. (1994). Suicidality and 5-hydroxyindoleacetic acid concentration associated with a tryptophan hydroxylase polymorphism. *Archives of General Psychiatry*, *51*, 34–38.
- Nielsen, D. A., Virkkunen, M., Lappalainen, J., Eggert, M., Brown, G. L., Long, J. C., et al. (1998). A tryptophan hydroxylase gene marker for suicidality and alcoholism. *Archives of General Psychiatry*, *55*, 593–602.
- Nolan, K. A., Volavka, J., Lachman, H. M., & Saito, T. (2000). An association between a polymorphism of the tryptophan hydroxylase gene and aggression in schizophrenia and schizoaffective disorder. *Psychiatric Genetics*, *10*, 109–115.
- Nomura, M., Andersson, S., Korach, K. S., Gustafsson, J. A., Pfaff, D. W., & Ogawa, S. (2006). Estrogen receptor-beta gene disruption potentiates estrogen-inducible aggression but not sexual behaviour in male mice. *European Journal of Neuroscience*, *23*, 1860–1868.
- Pillai, S. G., Chiano, M. N., White, N. J., Speer, M., Barnes, K. C., Carlsen, K., et al. (2006). A genome-wide search for linkage to asthma phenotypes in the genetics of asthma international network families: Evidence for a major susceptibility locus on chromosome 2p. *European Journal of Human Genetics*, *14*, 307–316.
- Reuter, M., & Hennig, J. (2005a). Pleiotropic effect of the TPH A779C polymorphism on nicotine dependence and personality. *American Journal of Medical Genetics B Neuropsychiatric Genetics*, *134*, 20–24.
- Reuter, M., & Hennig, J. (2005b). Association of the functional catechol-O-methyltransferase VAL158MET polymorphism with the personality trait of extraversion. *NeuroReport*, *16*, 1135–1138.
- Robins, D. M. (2005). Androgen receptor and molecular mechanisms of male-specific gene expression. *Novartis Foundation Symposium*, *268*, 42–52.
- Rujescu, D., Giegling, I., Bondy, B., Gietl, A., Zill, P., & Moller, H. J. (2002). Association of anger-related traits with SNPs in the TPH gene. *Molecular Psychiatry*, *7*, 1023–1029.
- Rujescu, D., Giegling, I., Gietl, A., Hartmann, A. M., & Moller, H. J. (2003). A functional single nucleotide polymorphism (V158M) in the COMT gene is associated with aggressive personality traits. *Biological Psychiatry*, *54*, 34–39.
- Rujescu, D., Giegling, I., Sato, T., Hartmann, A. M., & Moller, H. J. (2003). Genetic variations in tryptophan hydroxylase in suicidal behavior: Analysis and meta-analysis. *Biological Psychiatry*, *54*, 465–473.
- Sakai, J. T., Young, S. E., Stallings, M. C., Timberlake, D., Smolen, A., Stetler, G. L., et al. (2006). Case-control and within-family tests for an association between conduct disorder and 5HTTLPR. *American Journal of Medical Genetics B Neuropsychiatric Genetics*, *141*, 825–832.
- Sapolsky, R. M. (1991). Testicular function, social rank and personality among wild baboons. *Psychoneuroendocrinology*, *16*, 281–293.
- Setchell, J. M., & Dixon, A. F. (2001). Changes in the secondary sexual adornments of male mandrills (*Mandrillus sphinx*) are associated with gain and loss of alpha status. *Hormones & Behavior*, *39*, 177–184.
- Shih, J. C. (2004). Cloning, after cloning, knock-out mice, and physiological functions of MAO A and B. *Neurotoxicology*, *25*, 21–30.
- Smyth, D. J., Cooper, J. D., Bailey, R., Field, S., Burren, O., Smink, L. J., et al. (2006). A genome-wide association study of nonsynonymous SNPs identifies a type 1 diabetes locus in the interferon-induced helicase (IFIH1) region. *Nature Genetics*, *38*, 617–619.
- Staner, L., Uyanik, G., Correa, H., Tremereau, F., Monreal, J., Crocq, M. A., et al. (2002). A dimensional impulsive-aggressive phenotype is associated with the A218C polymorphism of the tryptophan hydroxylase gene: A pilot study in well-characterized impulsive inpatients. *American Journal of Medical Genetics*, *114*, 553–557.

- Strous, R. D., Bark, N., Parsia, S. S., Volavka, J., & Lachman, H. M. (1997). Analysis of a functional catechol-O-methyltransferase gene polymorphism in schizophrenia: Evidence for association with aggressive and antisocial behavior. *Psychiatry Research*, *69*, 71–77.
- Strous, R. D., Nolan, K. A., Lapidus, R., Diaz, L., Saito, T., & Lachman, H. M. (2003). Aggressive behavior in schizophrenia is associated with the low enzyme activity COMT polymorphism: A replication study. *American Journal of Medical Genetics B Neuropsychiatric Genetics*, *120*, 29–34.
- Trainor, B. C., Greiwe, K. M., & Nelson, R. J. (2006). Individual differences in estrogen receptor alpha in select brain nuclei are associated with individual differences in aggression. *Hormones and Behavior*, *50*, 338–345.
- Tsai, S. J., Wang, Y. C., Chen J. Y., & Hong, C. J. (2003). Allelic variants of the tryptophan hydroxylase (A218C) and serotonin 1B receptor (A-161T) and personality traits. *Neuropsychobiology*, *48*, 68–71.
- Volavka, J., Bilder, R., & Nolan, K. (2004). Catecholamines and aggression: The role of COMT and MAO polymorphisms. *Annals of the New York Academy of Sciences*, *1036*, 393–398.
- Westberg, L., Melke, J., Landen, M., Nilsson, S., Baghaei, F., Rosmond, R., et al. (2003). Association between a dinucleotide repeat polymorphism of the estrogen receptor alpha gene and personality traits in women. *Molecular Psychiatry*, *8*, 118–122.
- Widom, C. S., & Brzustowicz, L. M. (2006). MAOA and the “cycle of violence:” Childhood abuse and neglect, MAOA genotype, and risk for violent and antisocial behavior. *Biological Psychiatry*, *60*, 684–689.
- Zuckerman, M. (1984). Sensation seeking: A comparative approach to a human trait. *Behavioral and Brain Sciences*, *7*, 413–471.

Chapter 4

Constructing a Neurology of Anger

Michael Potegal and Gerhard Stemmler

Abstract In keeping with general neurological principles, earlier stage processing of verbal and visual triggers for anger must involve posterior and middle temporal cortices. As this processing typically evolves, it evokes memory of related events (e.g., past insults) and other relevant information from more anterior temporal areas. This mutual interaction shapes perceptions of anger-provoking challenges which activate cortical/subcortical circuits that prime and mediate angry/aggressive actions, e.g., cingulate motor areas 23 and 24 and medial/basal amygdala. The initial appraisals of anger in mid- and anterior temporal lobe are also transmitted anteriorly to ventromedial and orbitofrontal cortex. The latter integrates anger-provoking perceptions, e.g., combining the insulting verbal comment with the visual sneer, and weighs inhibitory factors like received or anticipated punishment, empathy with the offender and his relative social status. The combined result determines angry aggressive responses, if any, by disinhibiting the subcortical circuits activated by the temporal lobe. Interactions between ventromedial and orbitofrontal areas and/or feedback to the temporal lobe govern the escalation of aggression.

4.1 Overview of Anger in the Brain

For both practical and theoretical reasons, we begin the scientific chapters of this book with a discussion of what is known about the neurological bases of anger. On the clinical side, brain damage in the form of traumatic brain injury (TBI) in younger individuals and cerebral strokes and dementia in older people are common. Prevalence estimates suggest that about 5 million Americans live with some TBI-related disability (Thurman, Alverson, Dunn, Guerrero, & Snizek, 1999); 2.5 million have had strokes (Muntner, Garrett, Klag, & Coresh, 2002) and another 2.5 million suffer from dementia (Brookmeyer, Gray, & Kawas, 1998). Changes in emotion expression, typically in the direction of more frequent and intense anger, are often associated with such brain damage. After acute post-injury disturbance has resolved, residual agitation is reported in 5–71% of milder traumatic brain injury (TBI) and 31–71% of severe TBI (Kim, 2002; Tateno, Jorge, Robert, & Robinson, 2003). The wide range of rates is due to variation in site and nature of injury, nature of premorbid functioning, and many other variables. Among 40–80-year-old stroke patients (excluding those with severe aphasia), 32% showed increased trait anger 3–12 months following the event; in slightly

M. Potegal (✉)
University of Minnesota, Minneapolis, MN, USA
e-mail: poteg001@umn.edu

less than 25% of these the anger had no identifiable provocation (Kim, Choi, Kwon, & Seo, 2002). Epidemiological studies find agitation/aggression in 30% of people with dementia (Lyketsos, Lopez, & Jones, 2002). Conversely, Elliott (1982) found evidence of developmental or acquired brain dysfunction in 94% of 286 patients who had recurrent rages with little or no provocation. It would be clinically useful to be able to predict which patients will have such difficulties on the ward and after discharge from the hospital; eventually knowledge of brain mechanisms should also guide treatment.

On the theoretical side, all behavior starts with the brain; the study of neural mechanisms allows us to “carve (psychological) nature at its joints.” The neural bases of emotion are currently being revealed in unprecedented depth and detail by state-of-the-art methodologies such as single proton emission computed tomography (SPECT) and functional magnetic resonance imaging (fMRI). Affective neuroscience will develop most rapidly when physiological data are analyzed and interpreted in psychological context and physiological findings help shape psychological theories. In this spirit, we briefly and selectively review the elements of the neurology of anger as they are currently known and comment on how this knowledge sets the stage for the chapters that follow and relates to the issues raised by them.

The psychological complexity of anger revealed by the chapters in this book implies that its perceptual triggering, subjective experience, and overt display must involve linked neural circuits of several sorts. For example, if someone frowns and turns red with anger in response to a verbal insult, we can safely infer from general neurological knowledge that processing the language of the insult required the left posterior temporal/inferior parietal areas (at least if the individual were a right-handed male). The facial musculature was then activated through “extrapyramidal” (possibly dorsal tegmental) pathways to the seventh nerve, while the facial flushing was generated by hypothalamic activation transmitted through a sympathetic vasodilator pathway running from the second and third thoracic roots (Drummond & Lance, 1987). More generally, the clinical and experimental literature implicates the frontal and temporal lobes together with a descending set of subcortical structures in the mediation of anger. According to Golden et al.’s (1996) literature summary, the effects of damage to temporal and frontal lobes differ, with unwarranted, poorly directed anger being associated with temporal lobe involvement while, when the frontal lobes are impaired, the provocation is almost always clear, if trivial, and the response is usually directed toward that source. This chapter places these observations in the context of neurological first principles, i.e., the normal initial posterior-to-anterior flow of information in the brain. Accordingly, we start our review with the temporal lobe, then consider the amygdala within it and systems of associated subcortical structures. We note that these structures subserve various forms of aggression in other animals; they may mediate anger in humans, but mismatches in the current typologies of aggression across species limit the reconstruction of the subcortical neurology of human anger. We next address the role(s) of the frontal lobe, noting both feedback and feedforward effects from frontal to posterior regions, then conjecture about how these structures might dynamically function together to create the experience and expression of anger. We introduce some observations on epileptic seizures that are rarely considered in this context, but are quite relevant to it. We also review evidence for hemispheric asymmetries in the mediation of anger, adding some observations of our own (MP).

One important caveat in reviewing these clinical neurological studies is that they only rarely make the important distinction between anger and aggression that is repeatedly emphasized in this book. The mix of emotions and behaviors that is typically reported is presumptively equivalent to the anger–hostility–aggression (AHA) syndrome that was identified by Spielberger and is the focus of commentary by several other biologically focused chapters in this book, e.g., those by Reuter, by Bond and Wingrove, and by Williams. The selective and qualitative survey that follows includes studies in which “aggression” was reported but which can be reasonably assumed to have involved the AHA complex. We do not cover the burgeoning study of the brain mechanisms underlying

the perception of other people's anger, but note that some of the same areas that are involved in anger experience and expression, e.g., medial amygdala and lateral orbitofrontal cortex, appear to be involved in anger perception (Hermans, Ramsey, & van Honk, 2008; Esslen, Pascual-Marqui, Hell, Kochi, & Lehmann, 2004; for general review of the role of superior temporal gyrus and other areas in emotion perception, see Adolphs, 2002).

4.2 Temporal Lobes, Epilepsy, and Episodic Dyscontrol: Aggression or Anger?

A recent, small study found a correlation between changes in temporal lobe EEG activity and the intensity of recalled anger in presumably normal college students (Foster & Harrison, 2002). More extensive evidence, going back decades, implicates temporal lobe dysfunction in AHA in a number of patient and/or criminal populations. For example, Tonkonogy (1991) found that 5 of 14 ragefully violent individuals presenting with psychosis or seizures had gross abnormalities of the anterior-inferior temporal lobe as revealed by CT scans. Later and methodologically more adequate SPECT imaging detected basotemporal and orbitofrontal hypoperfusion in TBI patients with post-acute aggression (Greve et al., 2001). Unilateral anterior temporal (Hirono et al., 2000) or bilateral mid-temporal hypoperfusion (Lancôt et al., 2004) were found in Alzheimer patients who were aggressive relative to those who were not. According to Bufkin and Luttrell's (2005) review, 7 of 10 neuroimaging studies of aggressive and/or violent individuals reported temporal lobe dysfunction.

Increased AHA in individuals with temporal lobe epilepsy (TLE) was noted more than 50 years ago (Rey, Pond, & Evans, 1949). In Elliot's (1982) series, 30% of patients with recurrent, inexplicable anger were found to have complex partial seizures. TLE remains a continuing, if controversial, source of data. Although a few cases of anger expression during a temporal lobe seizure have been documented on video (cf., Trimble & van Elst, 1999), there is general agreement that ictal anger is rare in TLE; the emotion most typically associated with temporal lobe seizures is fear. Post-ictal struggling against restraint sometimes involves screaming and kicking (e.g., Yankovsky, Veilleux, Dubeau, & Andermann, 2005). The major finding of interest is that the frequency of *interictal* outbursts is elevated in TLE. Some of these arise out of an increased irritability that is part of an interictal syndrome of mood disorder and depression occurring in 19–67% of individuals with TLE (earlier studies focusing on personality found few psychopathological differences between TLE vs. non-temporal epilepsies, more recent studies focusing on mood dysregulation have found an increased prevalence in TLE, Glosser, Zvil, Glosser, O'Connor, & Sperling, 2000; Matsuura et al., 2003; Kanner, 2003). In some cases, as described by Blumer (2000) and others, irritability increases over hours or days preceding a TLE seizure; the irritability resolves after the seizure. This phenomenon has been documented in a systematic catalogue of TLE prodromae (Adamec, 1990), for review of earlier studies, see Potegal, 1994). Sometimes this intermittent irritability becomes a rage, triggered by only minimal provocation, that can last up to a few hours. This pattern has been called "episodic dyscontrol," appears in the Diagnostic and Statistical Manual – 4th Edition as Intermittent Explosive Disorder (ICD-9 312.24) and can arise from a number of different etiologies (Chapter 27 by R.W. Novaco, this book). Causality is suggested by the observation that temporal lobectomy in TLE patients with outbursts not only reduces seizures, but often ameliorates the rages as well (Fenwick, 1989, cf., Mpakopoulou, Gatos, Brotis, Paterakis, & Fountas, 2008 for similar claims about the beneficial effects of amygdalotomy on both seizures and aggression).

Of particular interest in the present context, Blumer (2000) clearly distinguishes between anger and aggression, noting that TLE rages remain verbal, are directed only against inanimate objects,

injury to others is carefully avoided, and the rage is followed by genuine remorse. “The highly emotional paroxysms of anger to the point of rage among patients with epilepsy lack the character of cold-blooded aggression” (Blumer, 2000, p 16). Of even greater interest are TLE patients whose dramatic angry/aggressive outbursts stand in stark contrast to their generally pleasant and well-controlled demeanor (see Potegal, 1994 for review). Again according to Blumer (2000), these outbursts rarely occur in physicians’ offices and families will not report them in the presence of the patient. Because the physician must know to ask about them, the occurrence of these starkly contrasting rages is not as widely recognized as might otherwise be the case.

To the extent that the general pattern is for patients’ anger, more than their aggression, to be accentuated in TLE, it is unsurprising that large scale, epidemiological surveys of patients with epilepsy failed to find an association between TLE and aggression (e.g., Brandt, Seidman, & Kohl, 1985; for review see Whitman, King, & Cohen, 1986). Nonetheless, the criticism that anger, aggression, or related problems may be due to the inimical life conditions that often co-occur with epilepsy, such as lower SES, as well the effects of anti-epileptic drugs, always needs to be taken into account. In any event, evidence for a temporal lobe involvement in anger continues to accumulate (e.g., Tebartz van Elst et al., 2000).

4.3 Hemispheric Asymmetries in Temporal Lobe Function

Evidence for cerebral asymmetries in emotion expression goes back at least as far as the reports of differing affective profiles following right vs. left strokes that appeared in the early 1970s. According to Bufkin & Luttrell (2005), 6 of 7 studies of neurological abnormalities in aggressive and/or violent groups involved reductions in left temporal lobe activity and/or excessive activity in right subcortical structures. SPECT studies of patients with dementia found heightened aggression associated with unilateral temporal hypoperfusion in left anterior (Hirono et al., 2000) and right mid-lobe (Lancôt et al., 2004) regions. Although the data are also mixed for TLE, there is a trend for greater left temporal involvement in the AHA syndrome in this condition, too (e.g., Tebartz van Elst et al., 2000).

As always, there are reservations about inferring the nature of normal function from the study of pathology. Complementing the evidence for asymmetric hemispheric involvement in the AHA behavior of adults with neuropathology are observations on children’s tantrums. Tantrums are of interest in this context because are the normative developmental prototype of anger, involving impressive displays of visibly angry behaviors in which many, if not most, 2- and 3-year olds intermittently stamp, throw things, shout, scream, hit, and kick. With simultaneous and increasing brain maturation and socialization, these immature and primitive “venting” displays are typically reduced by a lessening in the emotion of anger, a muting of the displays that accompany it, and replacement by more adaptive behavioral strategies. In Wranik and Scherer’s terms (Chapter 15, this book) “venting” anger is replaced by constructive anger (but perhaps by “malicious anger” in the cases of older and more hostile and vengeful children). The developmental exceptions to the stabilizing effects of maturation and socialization are children with high levels of negative affect and low self-regulation whose excessive tantrums persist into later childhood and beyond. In the extreme, children with clinical levels of psychopathology (e.g., juvenile bipolar disorder, obsessive–compulsive disorder) are infamous for their prolonged and intensely angry tantrums (e.g., Mick, Spencer, Wozniak, & Biederman, 2005). Thus, tantrums provide an opportunity to study anger and other intense emotions in a naturalistic setting.

A preliminary study comparing EEG asymmetries in tantrum-prone (TP) vs. nontantrum-prone (NTP) preschoolers (Potegal, Goldsmith, Chapman, Senulis, & Davidson, 1998) showed right frontal

activation associated with sadness, in keeping with the literature (e.g., Davidson, Shackman, Jeffrey, & Maxwell, 2004). The novel findings were that left temporal EEG activation was consistently and significantly associated with children's anger, as indicated by direct comparison of TP and NTP groups, parental report, and facial expressions of anger elicited by mild provocation (see Fig. 4.1).

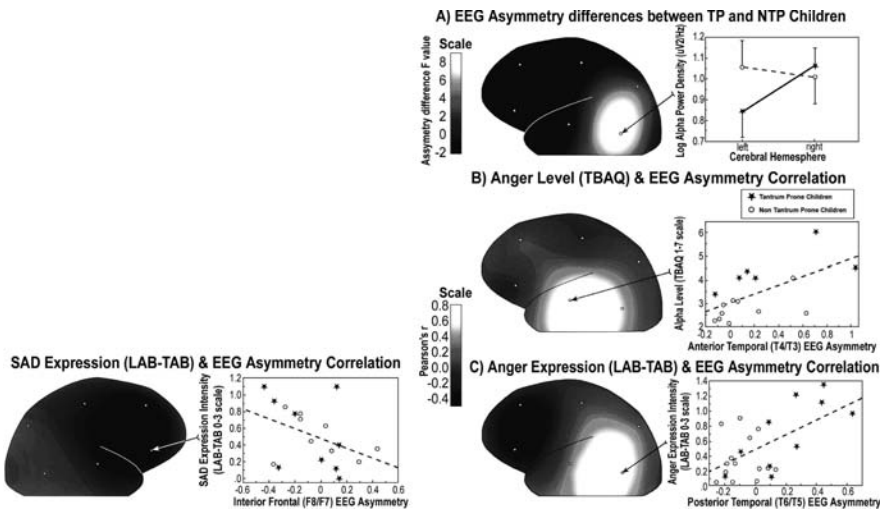


Fig. 4.1 Emotion-related EEG asymmetries in tantrum-prone (TP) and nontantrum-prone (NTP) children. Two telephone interviews about 14 months apart identified 10 consistently TP and 11 consistently NTP children (4 boys/group) among 230 Madison WI-area white, middle class families. Children were 41.4 ± 10.8 months old at first interview. When they were 48–63 months old, baseline EEG was recorded during a first laboratory session, temperament was evaluated during a second. The original parent-reported median tantrum frequency for TP children was 1–2/day, their median duration was 5–10 min; these values were at or above medians for age. These tantrums involved high anger (e.g., hitting, kicking, screaming) and distress. At the time of testing, median TP tantrum frequency was 3–6/week, median tantrum duration remained 5–10 min. Age- and sex-matched NTP children had either no tantrums or tantrums whose frequency (1–2/month) and duration (2–4 min) remained below the medians for age. Temperament evaluation with Goldsmith Reilly, Lemery, Longley, and Prescott (1994) LAB-TAB included videotaping four 1–5 min episodes involving mild restraint, toy loss, unfair candy sharing, or disappointment. In two of these, the mother was the source of the child's frustration, thus mimicking the typical tantrum situation (Eimon & Potegal, 1994). Parents also completed Goldsmith's (1996) Toddler Behavior Assessment Questionnaire (TBAQ). the TBAQ, TP children were more angry, active, and distractible than their NTP peers ($p < 0.006$). TP angry facial expressions in LAB-TAB episodes were more intense ($p < 0.05$). Topographic brain maps show the association of emotion measures with alpha band (10–12 Hz) power density ($\mu V^2/Hz$). Graphs associated with each map localize the lead showing the highest correlation of emotion measures with power density (panel A, right) or right/left hemisphere power ratios (EEG activation is inversely related to power; the higher the right/left ratio, the greater the left activation, Davidson 1988.) Across TP and NTP groups, sadness of facial expression was correlated with right frontal activation ($r = -0.41$, left lower panel), as in prior studies (e.g., Davidson et al. 2004). In regard to anger (right panels) there was: (a) greater activation asymmetry in left posterior temporal leads of TP vs. NTP children (T6/5 Group \times Hemi-sphere interaction $F(1,19) = 10.2$, $p < 0.005$) and across-group correlations between relative left temporal activation and anger in (b) parental report (T4/3, $r = 0.61$, $p < 0.02$) and (c) LAB-TAB angry facial expression (T6/5, $r = 0.57$, $p < 0.01$, Potegal et al., 1998.)

4.4 Subcortical Mediation of Anger in Humans and Its Putative Relationship with Several Forms of Aggression in Other Animals

Within the temporal lobe, the amygdala figures prominently in the neurology of anger. Clinical cases and series as well as reports of surgical and pharmacological interventions in several countries over many decades indicate that angry aggression in humans is associated with amygdala pathology. Most

recently, MRIs showed that 20% of TLE patients with aggression problems had severe atrophy of the amygdala (Tebartz van Elst et al. 2000). Fenwick's (1989) literature review noted that in patients whose angry aggression is associated with TLE, the aggression can be elicited by stimulating the amygdala. The medial amygdala may be particularly involved (Potegal, 1994). Conversely, since the first report by Narabayashi et al. in 1961 of bilateral amygdalotomy to manage severe aggressive behavior, more than 500 cases have been published in the scientific literature (Mpakopoulou et al., 2008). Although psychosurgery has been severely criticized, fallen into disrepute, and largely been replaced by psychopharmacological treatment, amygdalotomy for intractable aggression, most frequently aimed at the medial amygdala, continues to the present. Behavioral details in 13 studies reviewed by Mpakopoulou et al. (2008) were sketchy, but post-operative reductions of aggression were reported in 33–100% of cases (median improvement rate was 75% of patients); there were reportedly no effects on cognitive function or language in the majority of these cases. Only three studies reported long-term follow-up, but all three claimed sustained improvement at 2–9 years post-surgery.

Caudal to the amygdala, angry aggression is associated with hypothalamic dysfunction in the form of tumors (e.g., Tonkonogy & Geller, 1992, for review of case reports see Siegel, 2004, Table 5.1, pp. 117–118) and, more recently, hamartomas. Hamartomas are masses of disorganized but non-malignant tissue; they do not grow any faster than surrounding tissues, but can interfere with function. More than 75% of children whose hypothalamic hamartomas have grown to the point of causing gelastic seizures and general cognitive deterioration present with clinical levels of oppositionality, aggression, and rage (Weissenberger, Dell, & Liow, 2001; Fratelli, Liow, & Korenman, 2001). Conversely, older studies report that hypothalamotomy reduces angry aggression; these studies claim long-term improvement with relative specificity and few side effects (e.g., Sano & Mayanagi, 1988). More recent case reports assert clinically significant reduction of pathological aggression by stimulation in the posterior hypothalamic “triangle of Sano” (e.g., Franzini, Carlo Marras, Ferroli, Bugiani, & Broggi, 2005, but see Bejjani et al., 2002). Caudal to the hypothalamus, neuroimaging studies have found anger-associated changes in midbrain and anterior pons (Damasio et al., 2000).

These structures that have been identified individually in humans are actually elements of organized, caudally directed neuroanatomical systems for aggression that have been described in many species and carefully mapped in rodents and cats. Projections from specific nuclei in the amygdala descend through well-defined tracts to areas of hypothalamus that, in turn, project to specific sites in the midbrain and lower brainstem. The mapping of these systems in a number of laboratories over many years is a significant accomplishment in behavioral neuroscience (e.g., Adams, 2006; Kruk, 1991; Siegel, Roeling, Gregga, & Kruk, 1999). These neuroanatomical systems are best understood from the behavioral perspective of the now well-accepted distinctions among three types of species-typical aggression: predation, offense, and defense. Predatory aggression involves hunting behaviors, i.e., prey stalking, capture, and killing; the target is most often animals of other species. Offense includes dominance-motivated aggression initiated against conspecific territorial challengers or social rivals while defense refers to counterattack against predators or, more typically, conspecific attackers when escape is impossible (the “cornered rat” phenomenon). These behavioral systems differ in their evoking stimuli and sensory detection modalities, motivation, target, and topography of attack, hormone sensitivity, and other associated physiology. Their neuroanatomy also differs. Stimulation of cortical and medial amygdala nuclei facilitates defense but suppresses predatory attack; stimulation of central and lateral nuclei have opposite effects (Siegel et al., 1999). These effects are mediated through several pathways from the amygdala to the “attack zone” lateral to the ventromedial hypothalamic nucleus that has been thoroughly mapped in rats (Kruk, 1991). A classic example of behavioral differentiation of aggression systems in cats is the

silent, neck-biting, predatory attack elicited by lateral hypothalamic stimulation vs. the hissing, snarling, piloerected, back-arched affective defense elicited from medial hypothalamus (Siegel, 2004). The caudal extension of the defense system into the dorsal part of the midbrain periaqueductal gray has been thoroughly investigated (Adams, 2006; Siegel et al., 1999). In contrast, some observations suggest that the midbrain extension of offense involves the ventral tegmentum (Adams, 2006; Siegel et al., 1999). In turn, these areas project to lower brainstem structures that coordinate the motor and autonomic aspects of aggressive responding. If these more caudal structures are pathways of aggression, then the neuronal circuitry and patterns of activation that distinguish anger from other emotions and action patterns must lie in the cortex and upper limbic structures.

Overall, how does human anger and aggression map onto the offense/defense distinction? (Here we sidestep the misleading use of the term “predatory” to refer to what is actually offense-type aggression in humans). One view equates human anger with defensive aggression, the “fight” in Cannon’s fight or flight system. As defined by questionnaire studies, this form of aggression is triggered by immediate danger of physical harm when there is little chance of escape; it is unplanned, impulsive, and short lasting (seconds to minutes), its (non-conscious) goal is to reduce the immediate threat, it involves involuntary and stereotyped postures and behaviors, such as fist-clenching, obscenities, screaming, clawing, biting, and major autonomic arousal, and it can be accidentally redirected to unoffending others (Blanchard, Hynd, Minke, & Blanchard, 2001). This sort of desperate, fear-driven counterattack seems to correspond pretty well to “affective defense” in other animals. Interestingly, a reduction in right hypothalamic glucose utilization was found in alcohol-dependant batterers whose unpremeditated acts of violence were typically associated with a set of physical symptoms (e.g., palpitations, increased respiratory rate, tremor) and feelings of fear and/or being trapped; they were reportedly often remorseful afterward (George et al., 2004). Overall, however, such fear-driven attack may be relatively rare compared to the two main types of human aggression now generally recognized as reactive, irritable aggression vs. proactive, “instrumental,” aggression. Practically by definition, anger is associated with the former but not the latter (Chapter 14 by Hubbard et al., this book). So-called instrumental aggression is, in our view, predominantly dominance-related, but further discussion of this issue is beyond this chapter’s scope. Although challenged by some authors (e.g., Bushman & Anderson, 2001), the proactive/reactive dichotomy is both generally accepted in the field and helpful for clinical diagnosis and intervention (Merk, Bram Orobio de Castro, Koops, & Matthys, 2005; Hubbard, this book).

Some writers have identified reactive, angry aggression with a proposed category of “affective aggression” (e.g., Blair, 2004; Weinshenker & Siegel, 2002) in response to threat; Panksepp (1998) calls it the RAGE system. The major problem is that behaviors have been lumped by the “threat” stimuli that elicit them rather than by their own intrinsic characteristics. For other animals, the term “affective aggression” mistakenly lumps together the behaviorally very different categories of defense and offense. Defensive attack is a last resort in a hierarchy of fear-motivated behaviors (McNaughton & Corr, 2004). Offense does not involve fear and, in fact, fear abolishes offense (Blanchard & Blanchard, 1988). Defense is motivationally aversive, offense is positively reinforcing (under the right circumstances, Potegal, 1979) and so forth. For humans, the notion of an affective aggression in response to “threat” lumps together anger- and fear-driven behaviors. Anger and fear have different “core relational themes”; threats to authority or reputation are likely to elicit anger while those posing a risk to physical safety are more likely to elicit fear. Anger and fear have correspondingly different peripheral (Chapter 7 by Stemmler, this book) and central physiologies. “Affective aggression” appears to be a problematically mixed category for all species.

Overall, we argue that fear-driven, defensive aggression seems rather similar in humans and other animals, but it is not equivalent to human reactive aggression, which is anger-driven. Human anger

may be closer to offense than defense (Blanchard & Blanchard, 2003), but there are differences here, too. Anger is a negatively valenced emotion that many people try to avoid while offense is positively reinforcing (e.g., dominant animals will work for the opportunity to fight). Anger, like defensive aggression, involves strong sympathetic arousal, offense less so. Offense is strongly testosterone-related as is human dominance, anger much less so (e.g., Archer et al., 1998), and so on. In short, human anger does not correspond closely to any recognized form of aggression in other animals.

The medial amygdala is involved in offense in other animals and may be involved in anger in humans. Unlike the lateral amygdala, which has reciprocal projections to cortex and has expanded along with it in the evolution of the human brain, the medial amygdala has not grown. Given the importance of anger in human social interaction, these functions may have migrated to, or are shared with, other areas, such as ventromedial frontal cortex (VMFC) and orbitofrontal cortex (OFC). Perhaps as a consequence of this neuroanatomical transformation, anger as a derivative of offense has emerged as a uniquely human experience. This means we cannot automatically equate the neuroanatomical subsystems for one or another form of aggression in other animals with the neurocircuitry of anger in humans.

4.5 Roles of Frontal Lobe in Anger as Inferred from EEG and Lesion Data

From Phineas Gage on, large lesions that include ventral and medial frontal cortex have been understood to produce disinhibited and socially inappropriate behaviors, sometimes called “pseudopsychopathy” or “acquired sociopathy.” A multitude of clinical reports show such lesions can induce childish jocularity, boastfulness, verbal lewdness, egocentricity, and emotional lability (e.g., Silver & Yudofsky, 1987). Individuals with premorbid tendencies for AHA are the most likely to exhibit anger and aggression after frontal lobe damage further loosens control over these particular impulses (e.g., Greve et al., 2001; Tateno et al., 2003). The frontal lobe is one likely site of the genetic and neurochemical influences on the AHA syndrome discussed by Reuter (Chapter 3) and by Bond & Wingrove (Chapter 6), respectively, in this book.

4.5.1 Evidence from Seizures

Although not typically examined in the context of anger expression, the semiology and pathophysiology of some types of seizures are instructive. In particular, there is a class of frontally located “hypermotor” seizures that seems to include elements of angry behavior. Early case reports featured seizures that included whining, violent bimanual–bipedal thrashing, and screaming while prone (e.g., Yamanouchi, Noda, Sugai, Takashima, & Kurokawa, 1991). Angry facial expressions are occasionally seen (Rheims et al., 2008). Facial flushing preceding or during the onset of tantrums is typical, especially in 2-year olds (Potegal, 2000). In Manford et al.’s (1996) large-scale cluster analysis, about half of the “motor agitation” seizures started with flushing (as opposed to the pallor typically occurring with other seizure types). Roughly 80% of these seizures were of frontopolar/orbitofrontal origin (cf., Rheims Type 1 Hypermotor Seizures). Interictal hypometabolism associated with hypermotor seizures most frequently localizes to medial frontal and anterior cingulate gyrus (ACG) regions (e.g., Schlaug et al., 1997). Other evidence points to the ACG also being a part of this circuitry; it becomes activated in anger (Dougherty et al., 1999; Drexler et al., 2000) and cingulate seizures are reportedly associated with intense tantrums in adults (Mazars, 1970). This semiology is seen more rarely in temporal seizures; when it is, there is often reason to believe it

involves spread to frontal lobe (e.g., Wang, Mathews, Whetsell, & Abou-Khalil, 2008). We interpret the disorganized activation of angry behavior fragments in seizures of frontal origin to reflect control by the frontal lobe of acts that, under normal physiological circumstances, can be mobilized by anger into more coherent behavior.

4.5.2 Medial and Orbital Frontal Cortex: General Anatomy and Function

In the general flow of brain activity, perceptual information is transmitted anteriorly from the temporal lobe to the frontal lobe where action plans and motor responses are generated. The frontal lobe, in turn, selects from and enhances perceptual processing in posterior regions by return projections through the same tracts. Within the frontal lobe, many of the AHA–brain studies highlight ventromedial and orbital areas as the most likely substrates of anger and aggression. Here we sketch just enough of their functional neuroanatomy to generate a few testable hypotheses. Inconsistencies in nomenclature notwithstanding, VMFC is generally taken to include the medial aspect of the frontal pole, i.e., part of Brodmann’s areas 9 and 10 (BA9,10), and anterior cingulate gyrus (ACG). Many studies have shown that the “affective” portion of the ACG (BA32 and anteroventral BA24) is involved in the calculation of risks and benefits of actions associated with “hot” social–emotional challenges (e.g., Fukui et al., 2006), particularly, perhaps, those associated with response-related rewards (Elliot, Dolan, & Firth, 2000). Neuroanatomically, VMFC provides the major cortical output to visceromotor nuclei of the hypothalamus and brainstem (Öngür & Price, 2000). Phan, Wager, Taylor, and Liberzon’s (2002) review of 55 PET and fMRI studies of emotion found the medial prefrontal cortex to be activated in most emotions.

The ventral surface of the frontal lobe, the OFC, includes BA10 most anteriorly and BA47/12 most laterally; BA11 and BA13 form the anterior and posterior central zone, respectively. The medial OFC forms the angle of the frontal lobe where OFC and VMFC intermingle, this angle includes BA14. The OFC is neuroanatomically organized for the high-level integration of perceptual inputs from anterior temporal cortices and elsewhere via white matter tracts including the uncinate fasciculus (Öngür & Price, 2000). OFC and anterior temporal cortices are also richly interconnected with the amygdala. Factor analyses suggest that medial OFC is associated with reward-related processes while lateral OFC processes potential actions that might be associated with punishment and mediates response inhibition (Kringelbach & Rolls, 2004). More generally, the OFC is thought to calculate values of response outcomes in the context of current motivational state. The OFC and/or VMFC also mediate social information processing, such as empathy with others, as indicated by the deleterious effects of lesions in these regions on empathy-related affect recognition and theory-of-mind function (Shamay-Tsoory, Tomer, Berger, & Aharon-Peretz, 2003). Hurliman, Nagode, and Pardo (2005) found that the processing of exteroceptive information preferentially activated lateral OFC while the processing of interoceptive information preferentially activated medial OFC, consistent with their respective neuroanatomical networks. These authors reported reciprocal activation of medial and lateral OFC, i.e., increases in one area were accompanied by reductions in the other.

4.5.3 Medial and Orbital Frontal Cortex: Role in Anger and Aggression

A few studies of people with frontal lobe damage have distinguished between effects on anger and aggression. Grafman et al. (1996) obtained self and family member reports on Vietnam veterans 15 years after the war. Those with lesions that included anterior temporal lobe ($N=60$) were likely to

report more felt anger or hostility than was indicated by friends and relatives, but they were not different from non-head injured veteran controls in overt aggression as reported by observers. Veterans whose lesions included “medial frontal” cortex ($N=42$) were more frequently aggressive and violent by both self and family report, while those whose lesions included OFC ($N=28$) were reported to be the most aggressive and violent, even though they tended not to be as aware of these emotions and behaviors. Grafman, Vance, and Weingartner (1986) reported some hemispheric differences in anger-related frontal function. Veterans with right OFC lesions ($N=10$) reported themselves to be more angry and ready to fight, and also appeared more hostile, than those with left OFC lesions ($N=18$) or right “dorsolateral” lesions ($N=28$). Right OFC lesions were also associated with more psychopathology, changes in sexuality, and feelings of exhaustion. Veterans with left “dorsolateral” lesions ($N=17$) also self-reported high levels of anger and hostility. Dorsolateral frontal effects on anger and aggression would be unusual and these “dorsolateral” lesions may have included mesial/medial prefrontal cortex (Grafman, personal communication). As reviewed by Harmon-Jones et al. (Chapter 5, this book), the most consistent evidence for hemispheric asymmetries in the frontal lobe mediation of anger comes from EEG studies which show that greater left than right frontal activity is associated with anger-motivated approach while greater right than left frontal activity is associated with anger-motivated withdrawal. Cox and Harrison (2008) re-interpret some of these data, proposing that anger expression involves left frontal activation while anger inhibition involves right frontal activation.

The work of Grafman and colleagues has been updated by Rolls and collaborators in two studies of neurosurgical patients in the UK who had partial frontal lobectomies for tumors, aneurysms, or hemorrhage of anterior communicating or middle cerebral arteries. These studies included formal testing as well as self and family reports. Hornak et al.’s (2003) sample included 12 patients with unilateral or bilateral OFC lesions, 4 with unilateral lesions of the ACG/medial BA9 region (ACG/mBA9), and some with mixed or “other” (mostly dorsolateral) frontal lesions. Major changes in felt emotion were reported with either unilateral ACG/mBA9 or bilateral OFC lesions, with more increases than reductions in anger. [Consistent with this localization, trait anger was positively correlated with activation in subgenual ACG in the normal group of Goldstein et al.’s (2005) PET study.] Interestingly, the ACG/mBA9 and OFC lesions also reduced patients’ perception of other people’s vocal anger. Berlin, Rolls, and Kischka (2004) compared a larger group of OFC patients ($n=23$, 16 of whom had unilateral lesions) to those with other frontal lesions. OFC patients reported experiencing more anger and less happiness than either neurologically intact or lesion control groups, but there were no differences in sadness, fear, and disgust. In this study, anger and aggression within the OFC group were also correlated with generally disinhibited and inappropriate “frontal” behavior. A failure to develop appropriate social/emotional functioning, and/or an elevated AHA in adulthood, contrasts with the more normal development of other neurocognitive functions (e.g., language) in one case of agenesis of the frontal lobe (Ackerly, 1964) and two cases of orbital and lateral prefrontal cortex damage at or before 15 months of age (Anderson, Bechara, Damasio, Tranel, & Damasio, 1999). These reports suggest both a deep rooting of social/emotional function in the frontal lobe and a lack of developmental plasticity in the cortical regulation of anger.

Generalizing over these studies while sidestepping their inconsistencies, it appears that (1) VMFC and OFC are involved in the normal inhibitory control of felt emotions, (2) VMFC may participate in the processing of perceptions of self and/or other’s anger, but OFC seems to be more strongly involved in these functions, (3) OFC may be more specifically associated with anger (among the negative emotions) and aggressive behavior. In the context of current views of prefrontal cortical function, VMFC and OFC may mediate the effects of anger on the calculation of the potential payoffs and punishments for aggressive responding.

4.6 Reconciling Lesion and Neuroimaging Data

The foregoing lesion studies provide the most easily interpreted picture of OFC–VMFC inhibitory control of angry aggression. In general, neuroimaging studies of induced emotion support this localization but complicate the functional interpretation. Murphy, Nimmo-Smith, and Lawrence (2003) reviewed 106 PET and fMRI studies, among them nine studies which included anger. The authors included only research that used matched control conditions such that the difference between emotion and control conditions mostly reflected emotion-without-context effects (Stemmler, 1992). Considerable support was found for an affect program account of emotion, because fear, disgust, and anger had unique activation distributions. In anger, the most consistently activated region was the lateral orbitofrontal cortex (LOFC); in fear, the amygdala; in disgust, the insula/operculum and the globus pallidum (see Fig. 4.2) Two subsequent studies by Dougherty and colleagues confirm that anger recalled by control or healthy subjects is associated with increased rCBF in what appears to be an anterior locus within the left VMFC/OFC angle (Dougherty et al., 2004; Marci, Glick, Loh, & Dougherty, 2007). The latter study also included identification of increased sympathetic and decreased parasympathetic autonomic activity which is characteristic of anger as well as a differentiation from the cerebral rCBF patterns associated with happiness and sadness. In summary, neuroimaging confirms that OFC seems to be specifically associated with anger rather than with all negative and highly arousing emotions. It further suggests that lateral OFC may specifically regulate aggressive behavior.

In either case, interpretation is complicated by the expectation arising from the lesion data that increased anger and/or aggression would be associated with a *reduction* of frontal activation. Pietrini, Guazzelli, Basso, Jaffe, and Grafman (2000) reported just such a result, but neuroimaging generally

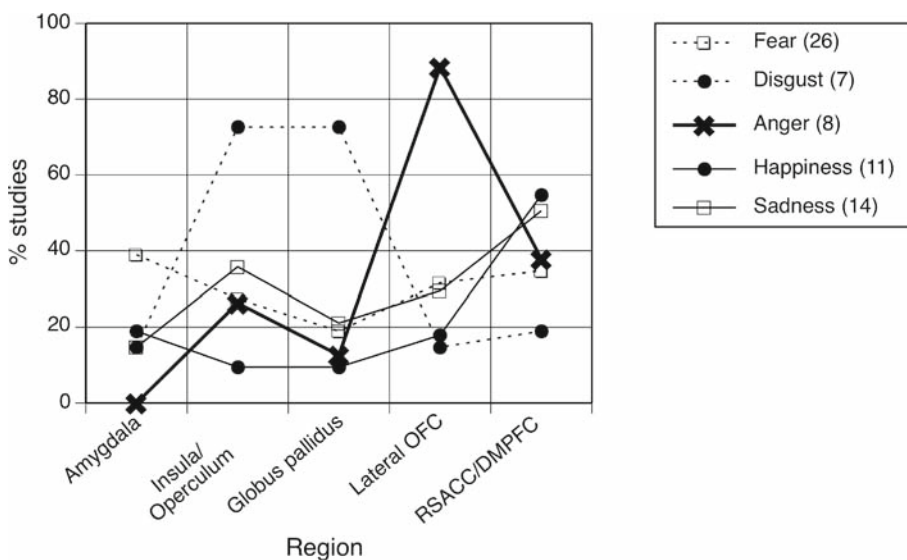


Fig. 4.2 Brain areas activated during emotions. Meta-analysis results of Murphy et al. (2003) on fMRI studies of emotion. Number of emotion-specific studies in *brackets*. Anger specifically evokes activity in the lateral OFC. OFC = orbitofrontal cortex, RSACC = rostral supracallosal anterior cingulate cortex, DMPFC = dorsomedial prefrontal cortex

reveals increases in rCBF, glucose utilization, etc. associated with anger. Two solutions may be proposed, neither of which is entirely satisfactory. Psychologically, one might suppose that, in the face of increased provocation, activation in these areas reflects a heightened inhibition of impulses to act out the felt anger (Dougherty et al. 1999; Lotze, Veit, Anders, & Birbaumer, 2007). While this argument may make sense for aggressive behavior (subjects in emotion-induction studies do not routinely become aggressive) it makes less sense for emotion, because these subjects do, in fact, become angry. An alternative, physiological explanation starts with the assumption that, absent provocation, tonic OFC output inhibits angry aggression. Cortical output is typically glutaminergic and excitatory, so this inhibition may involve inhibitory interneurons at target structure(s), e.g., the amygdala. OFC lesions reduce drive to these inhibitory neurons, yielding higher baselines of AHA. In intact individuals, provocation results in temporal lobe activation of GABAergic interneurons in OFC, which inhibits its output neurons, thus disinhibiting AHA. At the same time, cortical output to the striatum engages mesolimbic dopamine feedback loops that activate and modulate the cortical GABAergic interneurons (e.g., Steketee, 2003). The overall increase in cortical activation seen on neuroimaging reflects the dopaminergically (DA) amplified activity of these inhibitory neurons which, metabolically speaking, more than makes up for the reduced activity of the output neurons. This involvement of mesolimbic pathways can account for the (expected) reduction in anger-related rCBF in OFC observed in two studies of recalled anger in cocaine-dependant subjects (Drexler et al. 2000; Goldstein et al. 2005). The hypoactive DA pathways of these subjects do not modulate the cortical inhibition, and the observed decline of rCBF in their OFCs reflects an unmasking of the reduced activity in their OFC output neurons. We present this highly conjectural model to point out that the discrepancy between lesion and neuroimaging data is both a challenge that must be met and an opportunity to understand the complex neural circuitry of anger.

4.7 A Neuroanatomical Conjecture About the Dynamics of Anger

How might the foregoing neuroanatomy explain the typically rapid rise and slower fall of anger (Chapter 22 by Potegal, this book)? In keeping with general neurological principles, earlier stage processing of verbal and visual triggers for anger must involve posterior and middle temporal cortices. As this processing evolves, it evokes memory of related events (e.g., past insults) and other relevant information from more anterior temporal areas (Miyashita, 2004). This mutual interaction shapes preliminary perceptions of an anger-provoking challenge which activate cortical and subcortical circuits that prime and mediate angry/aggressive actions, e.g., cingulate motor areas 23 and 24 and medial/basal amygdala. Such direct pathways, from the temporal lobe to somato- and visceromotor brain structures without passage through the frontal lobe, are logically required because angry/aggressive acts are committed (even more) in the physical absence of the relevant frontal cortical areas. In fact, such pathways do exist (e.g., Morecraft & Van Hoesen, 1998; Stefanacci & Amaral, 2002).

The initial appraisals of anger in mid and anterior temporal lobe are rapidly transmitted anteriorly through the uncinate and cingulate fasciculi to VMFC and OFC. At least three studies showing that seizures initiated in the temporal lobe typically propagate along the usual white matter pathways (Spencer, 1988) to ipsilateral OFC are consistent with this idea (Lieb, Dashieff, & Engel, 1991; Saint-Hilaire & Lee, 2000; Weiser et al. 1980). In this context, we note a report of repeated immediate post-ictal aggression (screaming, kicking, and fighting restraints) in a patient whose seizures arose from periventricular, nodular heterotopia within the lateral aspect of both

temporal horns (Yankovsky et al., 2005). These authors comment with seeming surprise that, although the epileptogenic foci were located in the temporal lobe, the first change in scalp EEG occurred over the left frontal area shortly after seizure onset; it was then followed by bilateral frontal spreading (see also Tassinari et al., 2005). While the hypothesized anger-exciting interaction of perceptual and memory processes in the temporal lobe resembles the neo-associationistic network of Berkowitz (Chapter 16, this book), the putative successive activation of temporal then frontal areas seems more consistent with the sequential processing proposed in both appraisal models of anger (Chapter 15 by Wranik & Scherer, this book) and social information processing models of aggression noted by Lemerise (this book).

OFC functional neuroanatomy suggests that its role in the present context is to integrate anger-provoking perceptions forwarded from the temporal lobe, e.g., combining the insulting verbal comment with the visual sneer. However, something very important is missing from the strictly neuroanatomically based picture. What is missing is that the endpoint of the highest-level perceptual analysis and interpretation must, at the same time, be a start point for generating response strategies. We agree with Berkowitz (Chapter 16, this book) that the experience of anger incorporates impulses to aggressive action. Laboratory observations that anger is accompanied by jaw-clenching (Hutchinson, Pierce, Emley, Proni, & Sauer, 1977) suggest that, at its primitive core, anger potentiates the motor reaction of biting. Tassinari et al. (2005) report 11 patients (out of >1,000) in whom biting could be elicited by a visual or tactile stimulus near or on the face during a seizure. This response was associated with other fragmentary acts/gestures suggesting anger and/or aggression. Notably, this reflex-like response only occurred when the seizure involved both temporal and frontal regions simultaneously. In a single striking case report, a coherence analysis of surface and depth EEG recordings during seizures revealed a first phase of amygdala and anterior temporal co-activation followed by a second phase of greater amygdala and OFC coherence. The second phase involved intense affect, including anger and the impulse to bite (Bartomeil et al. 2002). These observations suggest that initial temporal to frontal transmission followed by temporofrontal coordination and interaction is integral to the full experience and expression of anger. It further supports the notion that it is the evocation of impulses to angry/aggressive action that uniquely characterizes the central motivational state of anger (Chapter 12 by Potegal & Qiu, this book).

As indicated above, we have not reviewed the neural circuitry underlying the perception of other people's anger, but did note the involvement of some of the same areas under discussion. Given our supposition that a principal function of the OFC is to engender impulses to action, including facial expressions, it is noteworthy that OFC damage also interferes with the perception of anger. Conversely, imitation of an angry expression specifically activated several areas, including parts of the medial frontal pole (BA10) and VMFC (BA11), whereas facial mimicry per se entailed emotion-unspecific activation of the right inferior PFC (Lee, Josephs, Dolan, & Critchley, 2006). The involvement of this action-related area suggests that subthreshold activation of motor programs for one's own angry facial expressions may play a role in forming a perception of other's anger (Adelmann & Zajonc, 1989). Such ideas, once part of the motor theory of perception, are now gaining more attention as examples of "embodiment" (Chapter 9 by Green et al., this book).

The findings that increases in angry aggression are most consistently associated with OFC lesions imply that it exerts an inhibitory control of angry aggression and that reduction of OFC output disinhibits aggression. This conjecture is consistent with the general role of the frontal lobe in plan generation and response selection and execution. It is also consistent with other classical neurological release phenomena of the frontal lobe, e.g., the snout, forced grasp, and other infantile reflexes that return when frontal lobe function is severely disrupted. Somewhat more subtle, stimulus-bound

“utilization behavior” reflects more minor changes in this same direction (Archibald, Mateer, & Kerns, 2001). Supposedly, 65% of murderers show one or more of these or other frontal release signs (Blake, Pincus, & Buckner, 1995). In normal social actions and interactions, the OFC may mediate the aggression inhibitory effects of punishment associated with potential aggressive actions, empathy with the potential target of aggression (Lotze et al., 2007), and, according to Blair’s (2004) innovative suggestion, rules of social conduct that modulate displays of anger and aggression according to relative social status. That is, the OFC may guide angry individuals to approach and confront an offender who is subordinate to them, but to retreat from and avoid an offender who is superior (e.g., Kuppens et al., 2004). Commentators as far back as Aristotle (Freese, 1939) and Aquinas (Reid, 2006) remarked that provocations that would elicit retaliatory aggression when coming from a low status offender may not even elicit anger when coming from a very high status individual. The mechanism of this could be inhibitory feedback from the OFC to temporal lobe that not only suppresses aggressive responding, but forestalls the experience of anger as well. By extension, the OFC is likely to coordinate with other frontal areas in selecting non-aggressive solutions to anger-provoking situations such as ignoring, problem-solving, or prosocial responding. Because the display rules of social conduct are likely to be encoded in procedural (non-declarative) memory, inhibitory control of aggression by the OFC may be mediated through the basal ganglia which are involved in this sort of memory. This might account for the anger-related activation sometimes seen in the striatum (Krämer, Jansma, Tempelmann, & Münte, 2007).

The increased aggression occurring with chronic OFC lesions might seem to suggest a simple loss of tonic inhibition, but this idea neglects frontotemporal interactions. Temporal lobe appraisals of anger that prime and potentially activate cortical/subcortical circuits for aggression are also transmitted to the OFC. In intact individuals with socially appropriate functioning, OFC processing of temporal input results in the selective gating of angry expressions and aggressive behavior appropriate to the moment. Anatomical loci for this hypothesized gating are likely to be behaviorally relevant regions in which temporal and frontal projections converge, e.g., the cingulate motor areas and medial/basal amygdala noted above. Because anger is dynamic, the relative timing of temporal activation and the coordinated partial frontal disinhibition would be crucial. Disruption of temporal–frontal white matter pathways might disturb this timing, leading to less effective damping of aggressive impulses by frontal cortex. Indeed, Tarkka et al. (2001) reported ERP evidence for a loss of sequential temporofrontal processing in violent vs. nonviolent alcoholics; recent diffusion tensor imaging studies suggest that disruption of temporofrontal white matter pathways is associated with behavioral dysregulation (Eluvathingal, Chugani, & Behen, 2006; Li, Mathews, Yang, Dunn, & Kronenberger, 2005). In cases of chronic frontal dysfunction, temporal lobe activation of aggression circuits is unregulated and results in overt aggression.

Brain lesion and neuroimaging data also implicate the VMFC in angry aggression. The output of VMFC to visceromotor nuclei of the hypothalamus and PAG strongly imply its involvement in the activation of the peripheral autonomic effects which contribute to the subjective sensations of anger (Chapter 7 by Stemmler and Chapter 10 by Koveces, this book). Case reports of two patients whose angry/aggressive outbursts were reduced by amygdalotomy noted concomitant reductions in autonomic arousal as indicated by post-operative reduction in skin conductance responses to arousing stimuli (Lee et al., 1998). The VMFC also evaluates response-related rewards (Kringelbach & Rolls, 2004), perhaps calculating the benefits of aggression in the presence of anger. These observations suggest that the VMFC may tend to facilitate aggression directly. Activation of dorsal VMFC (BA9) was correlated with revenge intensity in competition tasks that involved wins against, and losses to, opponents who delivered aversive finger pressure (Lotze et al., 2007) or noise (Krämer et al., 2007).

Krämer et al. (2007) conjectured that activation observed in the anterior insula might reflect the affective component of these tasks. However, integration of anger-focused and aggressive behavior-oriented research remains incomplete because these two studies reported no measures of experienced emotion or associated psychophysiology. Another effect likely to be related to ACG activation is the increased optimism about the outcome of action that is typically associated with anger (Chapter 17 by Litvak et al., this book). The ACG calculates risk and increased optimism signals a shift in its risk estimates.

Like the OFC, the VMFC projects back to temporal lobe. If VMFC indeed facilitates aggression, a positive feedback loop between VMFC and temporal lobe might account for the escalation seen in the rising phase of anger. In three patients whose visual hallucinations of violence were triggered by seizures arising in deep frontal areas, the OFC was suggested as the deep frontal source (Fornazzari, Farnik, Smith, Heasman and Ichise, 1992). Localizing EEG sources is always problematic and perhaps the VMFC was the source of this effect. OFC and VMFC are highly connected to the amygdala and changes in these particular feedback loops may play a role in pathological anger. In control subjects in Dougherty et al.'s (2004) PET study, left VMFC and amygdala were reciprocally activated, but in depressed patients with anger attacks this relationship was reversed so that VMPFC and amygdala were positively correlated. The suggestion that this reorganization predisposes to intense anger is consistent with the report that when the amygdala and OFC became more coherent during a seizure, the patient experienced anger and an impulse to bite (Bartomeil et al. 2002).

To the extent that the reported reciprocal activation of OFC and VMFC (Hurliman et al., 2005) applies to the instigation of anger, alternative or additional mechanisms of escalation could involve the VMFC becoming more dominant as anger increases and physiological reactions are activated. VMFC activation implies accentuation of the reinforcing and hedonically pleasurable aspects of aggression. VMFC activation would inhibit OFC, reducing consideration of aggression-related punishment (counterattack, revenge) thereby disinhibiting aggressive action. Importantly, OFC deactivation would also engender the loss of self-perception and disconnection from the environment, thereby providing a neuroanatomical substrate for the phenomenon of out-of-control anger (Chapter 22 by Potegal, this book). Fessler (Chapter 21, this book) suggests that a dramatic and extreme "male flash of anger" has evolved as an adaptation to forestall exploitation, intimidation, and attack by rivals and to enhance and protect reputation. If so, this response tendency could be instantiated by male brains that are tuned to operate in modes of frontotemporal positive feedback and/or OFC–VMFC reciprocal activation.

There are several recent reports of temporal and frontal lobe neuropathology co-occurring in aggression-prone individuals (Woerman et al. 2000; Juhasz, Behen, Muzik, Chugani, & Chugani, 2001). Reduced glucose metabolism in both areas in violent psychiatric patients (Volkow et al. 1995) is a similar co-occurrence. Neuropathology that occurs in both areas may increase the risk of AHA well over that associated with neuropathology in either area alone. Whether these areas can be simultaneously affected because of similar physiological vulnerabilities or because of a tight functional linkage remains to be determined.

Additional possible contributors to anger dynamics include corollary discharges arising from caudally directed, anger-related motor commands that would provide efference copy information to frontal and temporal areas through collaterals of their output pathways (cf., Bond, 1989). Finally, the demonstrated anger-enhancing effects of anger-related facial expressions; body postures; gestures and acts; and visceral activation (Chapter 22 by Potegal, this book) are likely to involve re-afferent somatic and autonomic feedback to temporofrontal circuits.

4.8 Implications for Future Psychological and Physiological Studies of Anger

The foregoing arguments imply that neuroimaging of subjects being provoked into anger should show sequential activation of temporal, then frontal areas. One reason why this has not yet been reported (to our knowledge) may be that the frontal areas associated with angry actions are more functionally focused and differentiated, and therefore more easily highlighted by the usual subtraction techniques used in neuroimaging, than are the temporal areas which process a diversity of anger and non-anger-related stimuli. Like some other anger-related characteristics (Chapter 18 by Schultze, this book), anger processing in the temporal lobe may be more detectable in people with high trait anger whose brains are especially reactive to real or imagined provocation.

Tracking the neural activity that accompanies the rise and fall of anger could provide crucial insights into the neurology of anger. This natural variation of anger over time is an ecologically appropriate context for comparing brain activation to peripheral psychophysiology and reported subjective experience. Experimental attention should also be paid to changes in the coherence and synchronization of frontal and temporal activity as markers of anger intensity.

One question of great importance for the theory of discrete emotions is whether there is a core pattern of neural activation associated with anger, independent of how it is induced. This question can be approached by studies of the same individuals in both competitive (reactive aggression) and recalled emotion paradigms. Such studies should correlate neuroimaging data with measures that include subjects' subjective estimates of the intensity of their anger (and other emotions), appropriate physiological measures (Chapter 7 by Stemmler, this book), and felt impulses to action (Roseman, Wiest, & Swartz, 1994). A relevant pair of predictions would be that impulse to action would be most closely correlated with OFC activity while autonomic measures might be more highly correlated with VMFC function. We also expect that different processes of anger reduction (e.g., decay, quenching, Chapter 22 by Potegal, this book) will involve different mechanisms impinging on the same central circuits. Note that statistical parameter mapping of such correlations, as we report here, may provide a different, and possibly more sensitive, localization of function than subtraction techniques.

4.9 Limitations and Caveats

One limitation of this short essay is that we do not consider the universe of genetic and environmental effects and confounds that so strongly influence whether and how people will experience or express anger. These include subcultural beliefs and practices, harsh upbringing, peer and gang influence, adult poverty, alcohol and drug use, and criminal history as well as role models of anger expression in the media in forms ranging from angry stand-up comics through hostile talk show hosts to violent videogames. All of these influences affect and act through the brain. Our conjectures suggest that much remains to be discovered about the functions of OFC, VMFC, and other brain structures in regard to anger. If the ideas presented here stimulate further investigation, this chapter will have accomplished its aim.

Acknowledgments Collection of the EEG data was supported by a grant to M. Potegal from the Harry Frank Guggenheim Foundation and by National Research Service Awards to M. Potegal from the National Institute for Neurological Disorders and Stroke (F33 NS09638) and the National Institute of Child Health and Human Development (F33 HD08208). At that time, the first author was a Fellow in the laboratory of Richard J. Davidson, where work was supported in part by an NIMH Center for Behavioral Sciences Research Grant (P50-MH52354) to the Wisconsin Center for Affective Neuroscience (R.J. Davidson, Director) and by an NIMH Research Scientist Award (KO5-MH00875).

References

- Ackerly, S. S. (1964). A case of paranatal bilateral frontal lobe defect observed for thirty years. In J. M. Warren & K. Akert (Eds.), *The frontal granular cortex and behavior* (pp. 192–218). New York: McGraw-Hill.
- Adams, D. B. (2006). Brain mechanisms of aggressive behavior: An updated review. *Neuroscience and biobehavioral reviews*, 30, 304–318.
- Adamec, R. E. (1990). Does Kindling model anything clinically relevant. *Biological Psychiatry*, 27, 249–279.
- Adelmann, P. K., & Zajonc, R. B. (1989). Facial efference and the experience of emotion. *Annual Review of Psychology*, 40, 249–280.
- Adolphs, R. (2002). Neural systems for recognizing emotion. *Current Opinion in Neurobiology*, 12, 169–177.
- Anderson, S. W., Bechara, A., Damasio, H., Tranel, D., & Damasio, A. R. (1999). Impairment of social and moral behavior related to early damage in human prefrontal cortex. *Nature Neuroscience*, 2, 1032–1037.
- John Archer, J., Biring, S. S., & Wu, F. C. W. (1998). The association between testosterone and aggression among young men: Empirical findings and a meta-analysis. *Aggressive Behavior*, 24, 411–420.
- Archibald, S. J., Mateer, C. A., & Kerns, K. A. (2001). Utilization behavior: Clinical manifestations and neurological mechanisms. *Neuropsychology Review*, 11, 117–130.
- Aristotle, XXII (1939). *The "art" of rhetoric* (J. H. Freese, Trans.). Cambridge: Harvard University Press.
- Bartolomeil, F., Guye, M., Wendling, F., Gavaret, M., Regis, J., & Chauvel, P. (2002). Fear, anger and compulsive behavior during seizure: Involvement of large scale fronto-temporal neural networks. *Epileptic Disorders*, 4, 235–241.
- Bejjani, B. P., Houeto, J. L., Hariz, M., Yelnik, J., Mesnage, V., Bonnet, A. M., et al. (2002). Aggressive behavior induced by intraoperative stimulation in the triangle of Sano. *Neurology*, 59, 1425–1427.
- Berlin, H. A., Rolls, E. T., & Kischka, U. (2004). Impulsivity, time perception, emotion and reinforcement sensitivity in patients with orbitofrontal cortex lesions. *Brain*, 127, 1108–1126.
- Blair, J. (2004). The roles of orbital frontal cortex in the modulation of antisocial behavior. *Brain and Cognition*, 55, 198–208.
- Blake, P. Y., Pincus, J. H., & Buckner, C. (1995). Neurologic abnormalities in murderers. *Neurology*, 45, 1641–1647.
- Blanchard, D. C., & Blanchard, R. J. (1988). Ethoexperimental approaches to the biology of emotion. *Annual Review of Psychology*, 39, 43–68.
- Blanchard, D. C., & Blanchard, R. J. (2003). What can animal aggression research tell us about human aggression?. *Hormones and Behavior*, 44, 171–177.
- Blanchard, D. C., Hynd, A. L., Minke, K. A., & Blanchard, R. J. (2001). Human defensive behaviors to threat scenarios show parallels to fear- and anxiety-related defense patterns of nonhuman mammals. *Neuroscience and biobehavioral reviews*, 25, 761–770.
- Blumer, D. (2000). Dysphoric disorders and paroxysmal affects: Recognition and treatment of epilepsy-related psychiatric disorders. *Harvard Review of Psychiatry*, 8, 8–17.
- Bond, A. B. (1989). Toward a resolution of the paradox of aggressive displays. II: Behavioral efference and the communication of intentions. *Ethology*, 81, pp. 235–249.
- Brandt, J., Seidman, L. J., & Kohl, D. (1985). Personality characteristics of epileptic patients: A controlled study of generalized and temporal lobe cases. *Journal of Clinical and Experimental Neuropsychology*, 7, 25–38.
- Brookmeyer, R., Gray, S., & Kawas, C. (1998). Projections of Alzheimer's disease in the united states and the public health impact of delaying disease onset. *American Journal of Public Health*, 88, 1337–1342.
- Bufkin, J. L., & Luttrell, V. E. (2005). Neuroimaging studies of aggressive and violent behavior. *Trauma, Violence and Abuse*, 6, 176–191.
- Bushman, B. J., & Anderson, C. A. (2001). Is it time to pull the plug on the hostile versus instrumental aggression dichotomy?. *Psychological Review*, 108, 273–279.
- Cox, D. E., & Harrison, D. W. (2008). Models of anger: Contributions from psychophysiology, neuropsychology and the cognitive behavioral perspective. *Brain Structure and Function*, 212, 371–385.
- Damasio, A. R., Grabowski, T. J., Bechara, A., Damasio, H., Ponto, L. L. B., Parvizi, J., et al. (2000). Subcortical and cortical brain activity during the feeling of self-generated emotions. *Nature Neuroscience*, 3, 1049–1056.
- Davidson, R. J. (1988). EEG measures of cerebral asymmetry: Conceptual and methodological issues. *International Journal of Neuroscience*, 39, 71–89.
- Davidson, R. J., Shackman, A. J., Jeffrey, S., & Maxwell, J. S. (2004). Asymmetries in face and brain related to emotion. *Trends in Cognitive Sciences*, 8, 389–391.
- Dougherty, D., Shin, L., Alpert, N., Pitman, R., Orr, S., Lasko, M., et al. (1999). Anger in healthy men: A PET study using script-driven imagery. *Biological Psychiatry*, 46, 466–472.

- Dougherty, D., Rauch, S. L., Deckersbach, T., Marci, C., Loh, R., Shin, L. M., et al. (2004). Ventromedial prefrontal cortex and amygdala dysfunction during an anger induction positron emission tomography study in patients with major depressive disorder with anger attacks. *Archives of General Psychiatry*, 61, 795–804.
- Drexler, K., Schweitzer, J. B., Quinn, C. K., Gross, R., Ely, T. D., Muhammad, F., et al. (2000). Neural activity related to anger in cocaine-dependent men: A possible link to violence and relapse. *American Journal of Addictions*, 9, 331–339.
- Drummond, P. D., & Lance, J. W. (1987). Facial flushing and sweating mediated by the sympathetic nervous system. *Brain*, 110, 793–803.
- Einon, D. F., & Potegal, M. (1994). Temper tantrums in young children. In M. Potegal & J. Knutson (Eds.), *Dynamics of aggression: Biological and social processes in dyads and groups* (pp. 157–194). Hillsdale, NJ: Erlbaum Assoc.
- Elliott, F. A. (1982). Neurological findings in adult minimal brain dysfunction and the dyscontrol syndrome. *Journal of Nervous and Mental Disease*, 170, 680–687.
- Elliot, R., Dolan, R. J., & Firth, C. D. (2000). Dissociable functions in the medial and lateral orbito frontal cortex: Evidence from human neuro imaging studies. *Cerebral cortex*, 10, 308–317.
- Eluvathingal, T. J., Chugani, H. T., Behen, M. E., et al. (2006). Abnormal brain connectivity in children after early severe socioemotional deprivation: A diffusion tensor imaging study. *Pediatrics*, 117, 2093–2100.
- Esslen, M., Pascual-Marqui, R. D., Hell, D., Kochi, K., & Lehmann, D. (2004). Brain areas and time course of emotional processing. *NeuroImage*, 21, 1189–1203.
- Fenwick, P. (1989). The nature and management of aggression in epilepsy. *Journal of Neuropsychiatry & Clinical Neurosciences*, 1, 418–425.
- Fornazzari, L., Farcnik, K., Smith, I., Heasman, G. A., & Ichise, M. (1992). Violent visual hallucinations and aggression in frontal lobe dysfunction: Clinical manifestations of deep orbitofrontal foci. *Journal of Neuropsychiatry and Clinical Neurosciences*, 4, 42–44.
- Foster, P. S., & Harrison, D. W. (2002). The relationship between magnitude of cerebral activation and intensity of emotional arousal. *International Journal of Neuroscience*, 112, 1463–1477.
- Franzini, A., Carlo Marras, C., Ferroli, P., Bugiani, O., & Broggi, G. (2005). Stimulation of the posterior hypothalamus for medically intractable impulsive and violent behavior. *Stereotactic and Functional Neurosurgery*, 83, 63–66.
- Fratelli, C. M., Liow, K., Korenman, L. M., et al. (2001). Cognitive deficits in children with hypothalamic hamartoma. *Neurology*, 57, 43–46, N=6/8 with screaming hit & kick.
- Fukui, H., Murai, T., Shinozaki, J., Aso, T., Fukuyama, H., Hayashi, T., et al. (2006). The neural basis of social tactics: An fMRI study. *NeuroImage*, 32, 913–920.
- George, D. T., Rawlings, R. R., Williams, W. A., Phillips, M. J., Fong, G., Kerich, M., et al. (2004). A select group of perpetrators of domestic violence: Evidence of decreased metabolism in the right hypothalamus and reduced relationships between cortical/subcortical brain structures in position emission tomography. *Psychiatry Research: Neuroimaging*, 130, 11–25.
- Glosser, G., Zwiil, A. S., Glosser, D. S., O'Connor, M. J., & Sperling, M. R. (2000). Psychiatric aspects of temporal lobe epilepsy before and after anterior temporal lobectomy. *Journal of Neurology, Neurosurgery, and Psychiatry*, 68, 53–58.
- Golden, C. J., Michele, L., Jackson, M. L., Peterson-Rohne, A., & Gontkovsky, S. T. (1996). Neuropsychological correlates of violence and aggression: A review of the clinical literature. *Aggression and Violent Behavior*, 1, 3–25.
- Goldsmith, H. H. (1996). Studying temperament via construction of the Toddler Behavior Assessment Questionnaire. *Child Development*, 67, 218–235.
- Goldsmith, H. H., Reilly, H. H., Lemery, K. S., Longley, S., & Prescott, A. (1994). *Manual for the preschool laboratory temperament assessment battery (Lab-TAB)*. Madison, WI: University of Wisconsin, Unpublished manuscript.
- Goldstein, R. Z., Alia-Klein, N., Leskovjan, A. C., Fowler, J. S., Wang, G. J., Gur, R. C., et al. (2005). Anger and depression in cocaine addiction: Association with the orbitofrontal cortex. *Psychiatry Research: Neuroimaging*, 138(1), 13–22.
- Grafman, J., Schwab, K., Warden, D., Pridgen, A., Brown, H. R., & Salazar, A. M. (1996). Frontal lobe injuries, violence and aggression: A report of the Vietnam Head Injury Study. *Neurology*, 46, 1231–1238.
- Grafman, J., Vance, S. C., Weingartner, H., et al. (1986). The effects of lateralized frontal lesions on mood regulation. *Brain*, 109, 1127–1148.
- Greve, K. W., Sherwin, E., Stanford, M. W., Mathias, C., Love, J., & Ramzinski, P. (2001). Personality and neurocognitive correlates of impulsive aggression in long-term survivors of severe traumatic brain injury. *Brain Injury*, 15, 255–262.
- Harmon-Jones, E., Sigelman, J. D., Bohlig, A., & Harmon-Jones, C. (2003). Anger, coping, and frontal cortical activity: The effect of coping potential on anger-induced left frontal activity. *Cognition and Emotion*, 17, 1–24.

- Hermans, E. J., Ramsey, N. F., & van Honk, J. (2008). Exogenous testosterone enhances responsiveness to social threat in the neural circuitry of social aggression in humans. *Biological Psychiatry*, 63, 263–270.
- Hirono, N., Mega, M., & Dinov, I. (2000). Left fronto temporal hypoperfusion is associated with aggression in patients with dementia. *Archives of Neurology*, 57, 861–866.
- Hornak, J., Bramham, J., Rolls, E. T., Morris, R. G., O'Doherty, J., Bullock, P. R., et al. (2003). Changes in emotion after circumscribed surgical lesions of the orbitofrontal and cingulate cortices. *Brain*, 126, 1691–1712.
- Hubble, G. (1997). Feminism and the battered woman: The limits of self-defense in the context of domestic violence. *Current Issues in Criminal Justice*, 9, 113–124.
- Hurliman, E., Nagode, J. C., & Pardo, J. V. (2005). Double dissociation of exteroceptive and interoceptive feedback systems in the orbital and ventromedial prefrontal cortex of humans. *The Journal of neuroscience*, 25, 4641–4648.
- Hutchinson, R. R., Pierce, G. E., Emley, G. S., Proni, T. J., & Sauer, R. A. (1977). The laboratory measurement of human anger. *Biobehavioral Reviews*, 1, 241–259.
- Juhász, C., Behen, M. E., Muzik, O., Chugani, D. C., & Chugani, H. T. (2001). Bilateral medial prefrontal and temporal neocortical hypometabolism in children with epilepsy and aggression. *Epilepsia*, 42, 991–1001.
- Kanner, A. M. (2003). Depression in epilepsy: Prevalence, clinical semiology, pathogenic mechanisms, and treatment. *Biological Psychiatry*, 54, 388–398.
- Kim, J. S. (2002). Agitation, aggression, and disinhibition syndromes after traumatic brain injury. *NeuroRehabilitation*, 17, 297–310.
- Kim, J. S., Choi, S., Kwon, S. U., & Seo, Y. S. (2002). Inability to control anger or aggression after stroke. *American Academy of Neurology*, 58, 1106–1108.
- Krämer, U. M., Jansma, H., Tempelmann, C., & Münte, T. F. (2007). Tit-for-tat: The neural basis of reactive aggression. *NeuroImage*, 38, 203–211.
- Kringelbach, M. L., & Rolls, E. T. (2004). The functional neuroanatomy of the human orbitofrontal cortex: Evidence from neuroimaging and neuropsychology. *Progress in Neurobiology*, 72, 341–372.
- Kuppens, P., Van Mechelen, I., & Meulders, M. (2004). Every cloud has a silver lining: Interpersonal and individual difference determinants of anger-related behaviors. *Personality and Social Psychology Bulletin*, 30, 1550–1564.
- Kruk, M. R. (1991). Ethology and pharmacology of hypothalamic aggression in the rat. *Neuroscience and biobehavioral reviews*, 15, 527–538.
- Laanctôt, K. L., Herrmann, N., Nadkarni, N. K., Leibovitch, F. S., Caldwell, C. B., & Black, S. E. (2004). Medial temporal hypoperfusion and aggression in Alzheimer disease. *Archives of Neurology*, 61, 1731–1737.
- Lee, G. P., Bechara, A., Adolphs, R., Arena, J., Meador, K. J., Loring, D. W., & Smith, J. R. (1998). Clinical and physiological effects of bilateral amygdalotomy for intractable aggression. *Journal of Neuropsychiatry and Clinical Neurosciences*, 10, 413–420.
- Lee, T. W., Josephs, O., Dolan, R. J., & Critchley, H. D. (2006). Imitating expressions: Emotion-specific neural substrates in facial mimicry. *Social Cognitive and Affective Neuroscience*, 1(2), 122–135.
- Li, T.-Q., Mathews, V. P., Yang, W., Dunn, D., Kronenberger, W. (2005). Adolescents with disruptive behavior disorder investigated using an optimized MR diffusion tensor imaging protocol. *Annals of the New York Academy of Sciences*, 1064, 184–192.
- Lieb, J. P., Dashieff, R. M., & Engel, J. (1991). Role of the frontal lobes in the propagation of mesial temporal seizures. *Epilepsia*, 32, 822–837.
- Lotze, M., Veit, R., Anders, S., & Birbaumer, N. (2007). Evidence for a different role of the ventral and dorsal medial prefrontal cortex for social reactive aggression: An interactive fMRI study. *NeuroImage*, 34, 470–478.
- Lyketsos, C. G., Lopez, O., Jones, B., et al. (2002). Prevalence of neuropsychiatric symptoms in dementia and mild cognitive impairment. *JAMA*, 288, 1475–1483.
- Manford, M., Fish, D. R., & Shorvon, S. D. (1996). An analysis of clinical seizure patterns and their localizing value in frontal and temporal epilepsies. *Brain*, 119, 17–40.
- Marci, C. D., Glick, D. M., Loh, R., & Dougherty, D. D. (2007). Autonomic and prefrontal cortex responses to autobiographical recall of emotions. *Cognitive, Affective, and Behavioral Neuroscience*, 7(3), 243–250.
- Matsuura, M., Oana, Y., Kato, M., Kawana, A., Kan, R., Kubota, H., et al. (2003). A multicenter study on the prevalence of psychiatric disorders among new referrals for epilepsy in Japan. *Epilepsia*, 44, 107–114.
- Mazars, G. (1970). Criteria for identifying cingulate epilepsies. *Epilepsia*, 11, 41–47.
- McCorkle, R. (1993). Living on the edge: Fear in a maximum-security prison. *Journal of Offender Rehabilitation*, 20 (1/2), 73–79.
- McNaughton, N., & Corr, P. J. (2004). A two-dimensional neuropsychology of defense: Fear/anxiety and defensive distance. *Neuroscience & Biobehavioral Reviews*, 28, 285–305.
- Merk, W., Bram Orobio de Castro, B., Koops, W., & Matthys, W. (2005). The distinction between reactive and proactive aggression: Utility for theory, diagnosis and treatment?. *European Journal of Developmental Psychology*, 2, 197–220.

- Mick, E., Spencer, T., Wozniak, J., & Biederman, J. (2005). Heterogeneity of irritability in attention-deficit/hyperactivity disorder subjects with and without mood disorders. *Biological Psychiatry*, 58, 576–582.
- Miyashita, Y. (2004). Cognitive memory: Cellular and network machineries and their top-down control. *Science*, 306, 435–440.
- Morecraft, R. J., & Van Hoesen, G. W. (1998). Convergence of limbic input to the cingulate motor cortex in the rhesus monkey. *Brain research bulletin*, 45, 209–232.
- Mpakopoulou, M., Gatos, M. H., Brotis, A., Paterakis, K. N., & Fountas, K. N. (2008). Stereotactic amygdalotomy in the management of severe aggressive behavioral disorders. *Neurosurgical Focus*, 25, E6, 1–6.
- Muntner, P., Garrett, E., Klag, M. J., & Coresh, J. (2002). Stroke prevalence between 1973 and 1991 in the US population 25 to 74 years of age. *Stroke*, 33, 1209–1213.
- Murphy, F. C., Nimmo-Smith, I., & Lawrence, A. D. (2003). Functional neuroanatomy of emotions: A meta-analysis. *Cognitive, Affective, and Behavioral Neuroscience*, 3, 207–233.
- Niedenthal, P. M., Barsalou, L. W., Winkelman, P., Krauth-Gruber, S., & Ric, F. (2005). Embodiment in attitudes, social perception, and emotion. *Personality and Social Psychology Review*, 9, 184–211.
- Öngür, D., & Price, J. L. (2000). The organization of networks within the orbital and medial prefrontal cortex of rats, monkeys and humans. *Cerebral Cortex*, 10, 206–219.
- Panksepp, J. (1998). *Affective neuroscience: The foundations of human and animal emotions* (pp. 187–205). Oxford: Oxford University Press, Chapter 10 Nature red in and claw: The Neurobiological Sources of Rage and Anger.
- Pietrini, P., Guazzelli, M., Basso, G., Jaffe, K., & Grafman, J. (2000). Neural correlates of imaginal aggressive behavior assessed by positron emission tomography in healthy subjects. *The American journal of psychiatry*, 157, 1772–1781.
- Phan, K. L., Wager, T., Taylor, S. F., & Liberzon, I. (2002). Functional neuroanatomy of emotion: A meta-analysis of emotion activation studies in PET and fMRI. *NeuroImage*, 16, 331–348.
- Potegal, M. (1979). The reinforcing value of several types of aggressive behavior: A review. *Aggressive Behavior*, 5, 353–373.
- Potegal, M. (1994). Aggressive arousal: The amygdala connection. In Potegal, M. & Knutson, J. (Ed.), *The dynamics of aggression: Biological and social processes in dyads and groups* (pp. 73–112). Hillsdale, NJ: Lawrence Erlbaum Associates.
- Potegal, M. (2000). Some characteristics, correlates, and consequences of visible autonomic activity in the temper tantrums of young children. In R. G. Barr, B. Hopkins, & J. A. Green (Ed.), *Crying as a sign, a symptom and a signal* (pp. 121–136). London: Mac Keith Press.
- Potegal, M., Goldsmith, H., Chapman, R., Senulis, J., & Davidson, R. (1998, July) Tantrums, temperament and temporal lobes. Poster presented at the biennial meeting of the International Society for Research on Aggression Ramapo NJ.
- Potegal, M., Kosorok, M. R., & Davidson, R. J. (1996). The time course of angry behavior in the temper tantrums of young children. *Annals of the New York Academy of Sciences*, 794, 31–45.
- Potegal, M., & Davidson, R. J. (2003). Temper tantrums in young children: 1) behavioral composition. *Journal of Developmental and Behavioral Pediatrics*, 24, 140–147.
- Potegal, M., Kosorok, M. R., & Davidson, R. (2003). Temper tantrums in young children: 2) tantrum duration and temporal organization. *Journal of Developmental and Behavioral Pediatrics*, 24, 148–154.
- Reid, J. P. (2006). *St Thomas Aquinas Summa Theologiae: 1a2ae. 40–48 Vol. 21 Fear and Anger*. Cambridge: Cambridge University Press.
- Rheims, S., Ryvlin, P., Scherer, C., Minotti, L., Hoffmann, D., Guenot, M., et al. (2008). Analysis of clinical patterns and underlying epileptogenic zones of hypermotor seizures. *Epilepsia*, 49, 1–11.
- Roseman, I. J., Wiest, C., & Swartz, T. S. (1994). Phenomenology, behaviors and goals differentiate discrete emotions. *Journal of Personality and Social Psychology*, 67, 206–221.
- Saint-Hilaire, J.-M., & Lee, M. A. (2000). Localizing and lateralizing value of epileptic symptoms in temporal lobe epilepsy. *Canadian Journal of Neurological Sciences*, 27(Suppl. 1), S1–S5.
- Sano, K., & Mayanagi, Y. (1988). Posteromedial hypothalamotomy in treatment of violent aggressive behaviour. *Acta Neurochirurgica Supplement*, 44, 145–151.
- Schlaug, G., Antke, C., Holthausen, H., Arnold, S., Ebner, A., Tuxhor, I., et al. (1997). Ictal motor signs and interictal regional cerebral hypometabolism. *Neurology*, 49, 341–350.
- Shamay-Tsoory, S. G., Tomer, R., Berger, B. D., & Aharon-Peretz, J. (2003). Characterization of empathy deficits following prefrontal brain damage: The role of the right ventromedial prefrontal cortex. *Journal of Cognitive Neuroscience*, 15, 324–337.
- Siegel, A. (2004). *The neurobiology of aggression and rage*. Boca Raton, FL: CRC Press.
- Siegel, A., Roeling, T. A. P., Gregga, T. R., & Kruk, M. R. (1999). Neuropharmacology of brain-stimulation-evoked aggression. *Neuroscience and biobehavioral reviews*, 23, 359–389.

- Silver, I. M., & Yudofsky, S. C. (1987). Aggressive behavior in patients with neuropsychiatric disorders. *Psychiatric Annals*, 17, 367–370.
- Spencer, S. (1988). Corpus callosum section and other disconnection procedures for medically intractable epilepsy. *Epilepsia*, 29(Suppl 2), S85–S99.
- Sprenkelmeyer, R., Rausch, M., Eysel, U. T., & Przuntek, H. (1998). Neural structures associated with recognition of facial expressions of basic emotions. *Proceedings: Biological Sciences*, 265, 1927–1931.
- Stefanacci, L., & Amaral, D. (2002). Some observations on cortical inputs to the macaque monkey amygdala: An anterograde tracing study. *Journal of Comparative Neurology*, 451, 301–323.
- Steketee, J. D. (2003). Neurotransmitter systems of the medial prefrontal cortex: potential role in sensitization to psychostimulants. *Brain Research Reviews*, 41, 203–228.
- Stemmler, G. (1992). The vagueness of specificity: Models of peripheral physiological emotion specificity in emotion theories and their experimental discriminability. *Journal of Psychophysiology*, 6(1), 17–28.
- Stemmler, G. (1997). Selective activation of traits: Boundary conditions for the activation of anger. *Personality and Individual Differences*, 22, 213–233.
- Stemmler, G., Heldmann, M., Cornelia, A. P., & Scherer, T. (2001). Constraints for emotion specificity in fear and anger: The context counts. *Psychophysiology*, 38, 275–291.
- Tarkka, I. M., Karhu, J., Kuikka, J., Pääkkönen, A., Bergström, K., Partanen, J., et al. (2001). Altered frontal lobe function suggested by source analysis of event-related potentials in impulsive violent alcoholics. *Alcohol and Alcoholism*, 36, 323–328.
- Tassinari, C. A., Tassi, L., Calandra-Buonaura, G., Stanzani-Maserati, M., Fini, N., Pizza, F., et al. (2005). Biting behavior, aggression, and seizures. *Epilepsia*, 46, 654–663.
- Tateno, A., Jorge, R. E., Robert, G., & Robinson, R. G. (2003). Clinical correlates of aggressive behavior after traumatic brain injury. *The Journal of Neuropsychiatry and Clinical Neurosciences*, 15, 155–160.
- Tebertz van Elst, L., Woermann, F. G., Lemieux, L., Thompson, P. J., & Trimble, M. R. (2000). Affective aggression in patients with temporal lobe epilepsy: A quantitative MRI study of the amygdala. *Brain*, 123, 234–243.
- Thurman, D. J., Alverson, C., Dunn, K. A., Guerrero, J., & Snizek, J. E. (1999). Traumatic brain injury in the United States: A public health perspective. *Journal of Head Trauma Rehabilitation*, 14, 602–615.
- Tiihonen, J., Kuikka, J. T., Bergström, K. A., et al. (1997). Single-photon emission tomography imaging of monoamine transporters in impulsive violent behaviour. *European Journal of Nuclear Medicine*, 24, 1253–1260.
- Tonkonogy, J. M. (1991). Violence and temporal lobe lesion: Head CT and MRI data. *The Journal of Neuropsychiatry and Clinical Neurosciences*, 3, 189–196.
- Tonkonogy, J. M., & Geller, J. L. (1992). Hypothalamic lesions and intermittent explosive disorder. *The Journal of Neuropsychiatry and Clinical Neurosciences*, 4, 45–50.
- Trimble, M. R., & Tebertz van Elst, L. (1999). On some clinical implications of the ventral striatum and the extended amygdala: Investigations of aggression. *Annals of the New York Academy of Sciences*, 877, 638–644.
- Volkow, N. D., Tancredib, L. R., Grant, C., Gillespie, H., Valentine, A., Mullani, N., et al. (1995). Brain glucose metabolism in violent psychiatric patients: A preliminary study. *Psychiatry Research: Neuroimaging*, 61, 243–253.
- Wang, L., Mathews, G. C., Whetsell, W. O., & Abou-Khalil, B. (2008). Hypermotor seizures in patients with temporal pole lesions. *Epilepsy Research*, 07, 005.
- Weinshenker, N. J., & Siegel, A. (2002). Bimodal classification of aggression: Affective defense and predatory attack. *Aggression and Violent Behavior*, 7, 237–250.
- Wieser, H. G., Meles, H. P., Bernoulli, C., & Seigfried, J. (1980). Clinical and chronotopographic psychomotor seizure patterns. *Acta Neurochirurgica Supplement*, 30, 103–112.
- Weissenberger, A., Dell, M., Liow, K., et al. (2001). Aggression and psychiatric comorbidity in children with hypothalamic hamartomas and their unaffected siblings. *Journal of the American Academy of Child and Adolescent Psychiatry*, 40(6), 696–703, N=12 83% ODD, 89% aggr.
- Whitman, S., King, L., & Cohen, R. (1986). *Epilepsy and violence: A scientific and social analysis psychopathology in epilepsy: Social dimensions*. New York: Oxford University Press.
- Woermann, F. G., van Elst, L. T., Koepp, M. J., Free, S. L., Thompson, P. J., Trimble, M. R., et al. (2000). Reduction of frontal neocortical grey matter associated with affective aggression in patients with temporal lobe epilepsy: An objective voxel by voxel analysis of automatically segmented MRI. *Journal of Neurology, Neurosurgery, and Psychiatry*, 68, 162–169.
- Yamanouchi, H., Noda, Y., Sugai, K., Takashima, S., & Kurokawa, T. (1991). Two cases of ictal automatism of frontal lobe origin. *No to Hattatsu (Brain and Development)*, 23, 492–496.
- Yankovsky, A. E., Veilleux, M., Dubeau, F., & Andermann, F. (2005). Post-ictal rage and aggression: A video-EEG study. *Epileptic Disorders*, 7, 143–147.

Chapter 5

Anger, Motivation, and Asymmetrical Frontal Cortical Activations

Eddie Harmon-Jones, Carly K. Peterson, and Cindy Harmon-Jones

Abstract This chapter reviews research on the motivational functions of anger and their association with asymmetric frontal cortical activations. In particular, anger is often associated with approach motivational inclinations, though certain individuals in certain situations may manifest anger that is associated with withdrawal motivational inclinations. Because anger is often associated with approach motivation, it is different from other negative emotions and thus is an emotion that permitted testing whether asymmetric (left vs. right) frontal cortical activity was due to positive vs. negative affect or approach vs. withdrawal motivation. Results of several studies revealed that anger is associated with greater relative left frontal activation. Moreover, manipulated increases in the approach motivation of anger cause even greater relative left frontal activation. These results support the idea that greater relative left frontal activity is associated with approach motivation and not positive affective valence. The chapter ends by discussing how this research challenges the idea that the frontal cortex is only involved in down-regulating negative emotions and by suggesting directions for future research examining connectivity among brain regions involved in anger and motivational processes.

Emotions are processes that involve involuntary action readiness (Frijda, 1986). Basic emotions, such as anger, provide organisms with relatively complex and biologically prepared behavioral potentials that assist in coping with major challenges to their welfare (Panksepp, 1998). However, these inherited behavioral tendencies exist only as potential ways of behaving in organisms with larger, more complex brains. Thus, although humans may possess the same emotional instincts as other animals, we may not be as controlled by the dictates of emotions and thus we have more choices (Panksepp, 1994). That is, our emotions can be regulated and thus may not directly affect behavior.

An emotion is not a “thing” but is best considered a psychological event that is made up of basic processes such as subjective feelings of pleasure or displeasure, facial-expression components, particular appraisals, and particular activation states and action plans (Frijda, 1993). Anger is a relatively unpleasant feeling, and it is described using words like *annoyed*, *angry*, and *enraged*, which in our view, express differences in intensity. Specific facial and vocal expressions of anger are

E. Harmon-Jones (✉)
Department of Psychology, Texas A&M University, College Station, TX, USA
e-mail: eddiehj@gmail.com

reviewed by Matsumoto et al. and by Green et al. in this book. Because humans are taught to control anger and its expression, facial and vocal anger vary in degree and because the social interactions evoking anger are complex, blends of emotions are the rule rather than the exception. Nevertheless, “on the face of an angry person there is almost always one or more of the innate components of the natural expression which signals his or her internal state” (Izard, 1977, p. 330).

Anger appears to serve a variety of adaptive functions. It organizes and regulates several psychological processes, such as self-defense and mastery. It also regulates social and interpersonal behaviors and organizes behaviors to assist with goal-directed action. However, because anger may give rise to maladaptive cognitions and behaviors, individuals and societies often attempt to regulate anger as a way of preventing intra- and interpersonal negative consequences, such as aggression. While there is some agreement about the functions of anger, the ways in which anger operates to serve these functions remain unclear. This chapter advances the following ideas: (1) Anger, unlike most negative emotions, shares some important commonalities with certain positive emotions; (2) anger often involves approach motivation; (3) but anger can also motivate withdrawal under certain circumstances; and (4) anger that motivates approach is associated with predominantly left frontal cortical activity, whereas anger that motivates withdrawal is associated with predominantly right frontal cortical activity.

5.1 Subjective Feelings and Anger

Consistent with other perspectives, this chapter proposes a conception of anger in which important commonalities override the “nuances” of individual anger experience or episodes (Berkowitz & Harmon-Jones, 2004a, 2004b; Shaver, Schwartz, Kirson, & O’Connor, 1987; Spielberger, Jacobs, Russell, & Crane, 1983; Spielberger, Reheiser, & Sydeman, 1995). While, for example, Averill (1982) proposed that annoyance and anger were different in kind, we share the view that anger ranges from low-level feelings such as irritation or annoyance to high-level feelings such as fury and rage, and these experiences reflect quantitative differences in the intensity of a single set of anger processes rather than qualitative differences across disparate sets of processes (see also Chapter 12 by M. Potegal & P. Qiu, this book).

Another commonality is that anger is most often experienced as a negative emotion. However, what is meant by negative is not always clearly defined in the literature. Emotions can be regarded as positive or negative (a) *because of the conditions that evoked the emotion*; (b) *because of the emotion’s adaptive consequences*; or (c) *because of the emotion’s subjective feel*.

Thus, the emotion of anger can be viewed as negative because it is evoked by aversive events. Most appraisal theorists regard the judgment of whether the emotion-evoking situation is positive or negative as the most frequent and most important way of distinguishing positive from negative emotions (Lazarus, 1991). On this view, anger is a negative emotion because it is evoked by unpleasant situations. Anger could also be viewed as either positive or negative according to its adaptive consequences. However, defining consequences as adaptive or maladaptive can be difficult (e.g., are the consequences in the short term or long term), and most emotions, even negative ones like fear, are adaptive.

Finally, anger could be viewed as either positive or negative by considering the subjective evaluation of the emotion, depending on whether an individual accepts or rejects, likes or dislikes the subjective experience of anger. In general, both state and trait studies examining the valence of anger indicate that most individuals regard anger as a negative experience. However, some people routinely find the experience of anger less negative than others (Harmon-Jones, 2004). In these studies, means on the 5-point 11-item “attitude toward anger” scale hover around 1.5, where 1 is strongly disagree

and 5 is strongly agree. Rarely do individuals score greater than 3.0, the mid-point of the scale, confirming that the experience of anger is typically negatively valenced. However, people who are frequently angry and in whose lives anger plays a major role routinely find the experience of anger less negative (Harmon-Jones, 2004). That is, the more trait anger people report on the Buss and Perry (1992) trait anger and hostility subscale (as measured by the Positive and Negative Affect Schedule – Expanded [PANAS-X], of Watson & Clark, 1991) the less negative or more positive they find the experience of anger to be, although the correlations are not so high as to suggest redundancy. These individual differences in attitude toward anger also relate negatively to trait fear (as measured by PANAS-X). Attitudes toward anger does not relate to self-reported affect intensity or social desirability. That some individuals find anger less negative than others may have important consequences for an individual's motivation to down-regulate anger or seek treatment for anger problems.

5.1.1 Relationship to Other Emotional Experiences

Anger is sometimes the primary or even sole emotional reaction to circumstances. However, anger often occurs amid other negative emotions, as many theoretical perspectives recognize (e.g., Berkowitz, 1989). In a recent experiment, self-reported affect was measured following interpersonal insult intended to elicit anger (Harmon-Jones, Vaugh-Scott, Mohr, Sigelman, & Harmon-Jones, 2004). In addition to reporting feeling more anger, participants reported feeling more active, alert, determined, proud, and strong than the no-insult control condition participants. These latter items are from the PANAS measure of activated positive affect (Watson, Clark, & Tellegen, 1988). On the surface, these results suggest that the insult manipulation caused more activated positive affect (PA). One methodological concern is that the words from the PA scale did not reflect positive feelings in this situation in which anger was present. However, these results for activated positive affect have since been replicated using different anger manipulations (Harmon-Jones, Harmon-Jones, Abramson, & Peterson, 2009). In these studies, anger and activated positive affect were positively correlated. However, statistically controlling for positive attitudes toward anger or positive emotions such as happiness does not undermine the PA–anger association (Harmon-Jones et al., 2009). In addition, within these studies, trait behavioral activation system (BAS; Gray, 1987) sensitivity positively related to both reported anger and reported activated positive affect, providing convergent evidence. Taken together, these results suggest that in addition to being associated with negative emotions, anger can also be associated with emotional experiences that have typically been thought of as positive (see Lerner et al., this book). Thus, positive affects that are high in approach motivational intensity might blend with anger, an approach motivated but negative affect, to cause increased anger responses and aggression. This blending of approach positive affect and anger may be experienced as a unitary feeling of being determined, strong, and angry.

Research has revealed that appetitive and consummatory components of reward processes relate to different experiences of positive affect in humans. This work has suggested that while seeking out and obtaining a reward, high-approach positive affect occurs, whereas consummatory responses after obtaining a reward cause satisfaction, a low-approach positive affect (Knutson & Wimmer, 2007). Neurobiological differences exist between pregoal and postgoal attainment positive affect, in the prefrontal cortex, nucleus accumbens, and other structures (Davidson, 1998; Davidson & Irwin, 1999; Knutson & Peterson, 2005; Knutson & Wimmer, 2007). As we will see, pregoal positive affect, which may assist in promoting goal-directed action, activates the same approach motivation neural circuitry involved in approach-oriented anger (Harmon-Jones, 2006; Peterson, Shackman, & Harmon-Jones, 2008).

5.2 Motivational Components of Anger

As noted above, a number of theorists have suggested that anger is an emotion that evokes behavioral tendencies of approach (e.g., Darwin, 1872/1965; Ekman & Friesen, 1975; Plutchik, 1980; Young, 1943). Of course, emotions are complex phenomena and discrete emotions may elicit both approach and withdrawal tendencies. Eckhardt and Deffenbacher's (1995) nine empirically derived categories of anger-related behaviors include refusal to cooperate and sullen withdrawal as well as various forms of verbal and physical aggression against property and people. Overall, however, we believe that the dominant behavioral tendency associated with anger is approach (i.e., movement toward the perceived source of the anger).

5.2.1 Behavioral and Subjective Evidence

Indeed, research has indicated that anger is often associated with attack (e.g., Berkowitz, 1993). Moreover, Depue and Iacono (1989) have suggested that irritable aggression is part of the behavioral facilitation system, a biobehavioral system similar to the behavioral activation system (BAS; Gray, 1987). Whether anger results in a general tendency to approach as compared to a specific tendency to aggress is currently a topic of debate with some suggesting the former (Lewis, 1993) and some the latter (Berkowitz, 1999).

In support of the idea of anger evoking approach motivation, studies on contingency learning by Lewis and colleagues (Lewis, Alessandri, & Sullivan, 1990; Lewis, Sullivan, Ramsey, & Alessandri, 1992) found that infants who displayed anger during extinction demonstrated the highest levels of joy, interest, and a required arm pull operant when the learning portion of the task was reinstated. Thus, subsequent to frustrating events, prior anger may maintain and increase task engagement and approach motivation.

In research with adults, Baron (1977) demonstrated that angry individuals are reinforced positively by signs of their tormentor's pain. Participants who had been deliberately provoked by another individual were given an opportunity to assault him in return. Indications that their first attacks were hurting their target led to increased aggression for previously provoked participants, but reduced aggression by participants who had not been provoked. The initial signs of their victim's suffering showed the angry subjects they were approaching their aggressive goal and thus evoked even stronger assaults from them. Other research is consistent with these findings (e.g., Berkowitz, Cochran, & Embree, 1981).

Additional support for the idea that anger is associated with approach motivation comes from research integrating the theory of reactance, a motivational state aimed at restoring control, with learned helplessness theory (Wortman & Brehm, 1975). According to this model, how individuals respond to uncontrollable outcomes depends on their evaluation of the importance of the outcome and their expectation of being able to control it. When individuals expect to be able to exercise control, the first few bouts in which they find that they cannot control the outcomes should arouse reactance. After several exposures to such uncontrollable outcomes, these individuals should become convinced that control is not in their power and they should show decreased motivation (i.e., learned helplessness). In other words, reactance will precede helplessness for individuals who initially expect control. In one study testing this model, individuals who exhibited angry feelings in response to one unsolvable problem had better performance and were presumably more approach motivated on a subsequent cognitive task than did participants who exhibited less anger (Mikulincer, 1988).

Other research has revealed that state anger relates to high levels of self-assurance, physical strength, and bravery (Izard, 1991), inclinations associated with approach motivation. Additionally, Lerner and Keltner (2001) found that anger (both trait and state) is associated with optimistic expectations, whereas fear is associated with pessimistic expectations. Moreover, happiness was associated with optimism, making anger and happiness appear more similar to each other in their relationship with optimism than are fear and anger. Although Lerner and Keltner interpreted their findings as being due to the appraisals associated with anger, it seems equally plausible that it was the approach motivational character of anger that caused the relationship of anger and optimism. That is, anger creates optimism because anger engages the approach motivational system and produces greater optimistic expectations.

Other evidence supporting the idea that anger is associated with an approach orientation comes from research on bipolar disorder. The emotions of euphoria and anger often occur during manic episodes of bipolar disorder (Cassidy, Forest, Murry, & Carroll, 1998; Depue & Iacono, 1989; Tyrer & Shopsin, 1982). Both euphoria and anger may be approach-oriented processes, and a dys-regulated or hyperactive approach system may underlie mania (Depue & Iacono, 1989; Fowles, 1993). Research suggests that hypomania/mania involves increased left frontal brain activity and approach motivational tendencies. In this work, it has been found that individuals who have suffered damage to the right frontal cortex are more likely to evidence mania (see review by Robinson & Downhill, 1995). This finding is consistent with the view that mania may be associated with increased left frontal activity and increased approach tendencies, because the approach motivation functions of the left frontal cortex are no longer restrained by the right hemisphere withdrawal system (see below). Furthermore, lithium carbonate, a treatment for bipolar disorder, reduces aggression (Malone, Delaney, Luebbert, Cater, & Campbell, 2000), suggesting that anger and aggression correlate with the other symptoms of bipolar disorder. In addition, trait anger has been found to relate to high levels of assertiveness and competitiveness (Buss & Perry, 1992).

Additional individual differences studies support the hypothesis that trait anger is related to trait approach motivation, or more specifically, to trait BAS sensitivity (Harmon-Jones, 2003). In two studies, trait BAS sensitivity, as assessed by Carver and White's (1994) scale, was positively related to trait anger, as assessed by the Buss and Perry (1992) aggression questionnaire. Additionally, in Study 2, BAS was positively correlated with physical aggression. Carver (2004) has also found that trait BAS predicts state anger in response to situational anger manipulations. These results support the hypothesis that anger is related to approach motivation.

5.3 Triggers of Anger

Researchers have often considered anger to be the result of physical or psychological restraint or of interference with goal-directed activity (Darwin, 1872/1965; Izard, 1977; Lewis, 1993). This action-oriented approach to understanding the cause of anger is consistent with the postulates of other major theoretical perspectives.

5.3.1 Reinforcement Approaches

Neo-behaviorists suggested that the actual or signaled arrival or termination of pleasant or unpleasant events (positive or negative reinforcers) was the primary cause of emotions (Mowrer, 1960). Gray (1987) extended these ideas by including stimulus omissions and interactions with individuals'

resources, such as ability to deal with events (see also Rolls, 1999). According to these models, angry emotions occur as a result of termination of a positive reinforcer or the omission of an expected one. Along these lines, Lewis (1993) proposed that the thwarting of a goal-directed action is an unlearned cause of anger. In one experiment, after 2- to 8-month-old infants were conditioned to move one of their arms in order to see a picture of another baby's smiling face, the infants were exposed to an extinction phase in which the arm movement no longer revealed the pleasing picture. This "frustrating" event caused the majority of the infants to exhibit angry facial expressions (Lewis et al., 1990).

5.3.2 Cognitive Neo-association Approach

In considering the causes of anger, Berkowitz (1989) extended the original frustration–aggression model (Dollard, Doob, Miller, Mowrer, & Sears, 1939) with a cognitive neo-associative model of anger and aggression. According to this model, any unpleasant situation, including pain, discomfort, frustration, or social stress, provokes negative affect. This negative affect is associated with fight-and-flight motivation. The individual's prior experiences have formed associations with situational cues that shape responses to the present circumstances. If these cues prompt escape tendencies and motivation, then the flight system is activated and the person experiences mostly fear. If the cues prompt attack tendencies and aggressive motivation, then the fight system is activated and he or she experiences mostly anger. Note that in this view, the emotions of fear or anger are generated in parallel with but are not causal to the behavioral tendencies (see Chapter 16 by Berkowitz, this book).

5.4 Neural Components of Anger

5.4.1 Motivational Direction: Approach and Withdrawal

Activation of a system of brain structures and neurochemicals are involved in anger and aggression, as described in Chapter 1 by M. Potegal and G. Stemmler (this volume) and Chapter 6 by A.J. Bond and J. Wingrove (this volume). Here, we focus on anger and asymmetrical frontal cortical activity. We start with basic biology. Research with organisms as simple as toads has revealed that approach and withdrawal processes are left and right lateralized (Vallortigara & Rogers, 2005). These lateralizations involve structures other than the frontal cortex, which amphibians lack. It is possible that sub-cortical structures are lateralized for approach and withdrawal motivational processes in amphibians, reptiles, and birds and that these lateralizations are preserved and elaborated into the frontal cortices of primates. The approach and withdrawal processes implemented by asymmetrical frontal cortices have been observed in rhesus monkeys (e.g., Kalin, Shelton, Davidson, & Kelley, 2001) and humans as early as 2–3 days of age (Fox & Davidson, 1986).

5.4.2 Valence: Positive and Negative Affect

Research conducted over the last 30 years with humans has revealed that the frontal lobes are asymmetrically involved in emotional and motivational processes. Interest in the relationship between asymmetrical frontal brain activity and emotional processes was sparked in part by systematic

observations that damage to the left frontal cortex caused depression, whereas damage to the right frontal cortex caused mania (Robinson, Kubos, Starr, Rao, & Price, 1984). Following closely after these observations, research demonstrated that both trait and state positive affect was associated with increased left frontal cortical activity, whereas trait and state negative affect was associated with increased right frontal cortical activity (see review by Silberman & Weingartner, 1986). Other studies revealed that approach-motivated behavior was associated with increased left frontal cortical activity, whereas withdrawal-oriented behavior was associated with increased right frontal cortical activity (Schiff & Bassel, 1996; Schiff, Guirguis, Kenwood, & Herman, 1998; Sobotka, Davidson, & Senulis, 1992).

Studies implicating asymmetrical frontal cortical activity in positive vs. negative affect have used a variety of neuroscience recording methods: positron emission tomography (PET; Thut et al., 1997), functional magnetic resonance imaging (fMRI; Canli, Desmond, Zhao, Glover, & Gabrieli, 1998), event-related brain potentials (ERPs; Cunningham, Espinet, DeYoung, & Zelazo, 2005; Peterson, Gable, & Harmon-Jones, 2008), and electroencephalographic activity (EEG; Coan & Allen, 2003). Clinical lesion studies (Robinson & Downhill, 1995) and experimental interventions (repetitive transcranial magnetic stimulation (rTMS) van Honk, Schutter, d'Alfonso, Kessels, & de Haan, 2002) also support these conclusions.

5.4.3 *Motivational Direction vs. Valence*

Until the late 1990s, all studies examining the relationship between asymmetrical frontal cortical activity and emotion had confounded affective valence (positive vs. negative affect) with motivational direction. That is, all positive affective states/traits (e.g., joy, interest) that had been empirically examined were approach motivating, whereas all negative affective states/traits (e.g., fear, disgust) were withdrawal motivating. To understand whether asymmetrical frontal cortical activations were due to affective valence or motivational direction, studies of an emotive state that avoided this confound of valence and motivational direction was needed. Because past research had suggested that anger is a negative emotion that evokes approach motivational action tendencies, Harmon-Jones and colleagues began investigating the relationship of anger to asymmetrical frontal cortical activity. If asymmetrical frontal cortical activity relates to motivational direction, then *anger should relate to greater left than right frontal activity*, because anger is associated with *approach motivation*. On the other hand, if asymmetrical frontal cortical activity relates to affective valence, then *anger should relate to greater right than left frontal activity*, because anger is associated with *negative valence*.

By investigating the relationship of anger with asymmetrical frontal cortical activity, these investigations were intended to provide a more complete understanding of the psychological and behavioral functions of asymmetrical frontal cortical activity. In addition, basic research on anger and its underlying neural systems can provide insights useful for understanding the relationship of motivational direction and affective valence. Most contemporary theories of emotion assume that positive affects are only related to approach motivation, whereas negative affects are only related to withdrawal motivation (Lang, 1995; Watson, 2000). Exploring anger can provide a better understanding of how these two important dimensions are related. Finally, by understanding basic processes involved in anger, we as a society should be in a better position to explain, predict, treat, and control anger when necessary.

Much of the research examining the neural systems involved in anger comes from studies that have utilized EEG activity to measure regional brain activation. Because of this, we provide a brief overview of EEG measurement.

5.4.4 EEG Measurement

The EEG observed at the human scalp is presumed to reflect postsynaptic dendritic potentials whose temporal and spatial organization allows them to be summed into a recordable signal. The extended durations of postsynaptic dendritic potentials, on the order of tens of milliseconds, allow them to be synchronized in time. Spatially, these potentials arise from the dendrites of layers of cortical neurons that are arranged/aligned in parallel in an “open field” structure (i.e., the dendrites all project outward from their neuronal cell bodies toward the scalp while their axons all project in and down from the cell bodies). The raw EEG signal is a complex waveform that can be analyzed in the temporal or frequency domains. Processing of the temporal aspect is typically done with event-related potential designs and analyses. Processing of the frequency aspect is done by decomposing the EEG signal using fast Fourier transforms (FFT). That is, multiple frequency bands can be extracted from the raw EEG by the FFT. Alpha frequency ranges from 8 to 13 Hz (cycles per s). Past research using a variety of other measures of cortical activation (Lindsley & Wicke, 1974), such as positron emission tomography (Cook, O’Hara, Uijtdehaage, Mandelkern, & Leuchter, 1998) and fMRI (Goldman, Stern, Engel, & Cohen, 2002), has suggested that alpha power is inversely related to cortical activation.

5.4.5 Anger and EEG Activity – Correlational Studies of Trait Anger

In the first study examining the relationship of anger and asymmetrical frontal cortical activity, Harmon-Jones and Allen (1998) found that trait anger related to increased left frontal activity and decreased right frontal activity during resting baseline. Trait anger was measured in college students using the Buss and Perry (1992) anger subscale of the Aggressive Questionnaire. EEG activity was acquired during a 6-min resting, baseline measurement session. Hewig, Hagemann, Seifer, Naumann, and Bartussek (2004) replicated these effects using the anger-out measure of the State–Trait Anger Expression Questionnaire (STAXI; Spielberger, 1988). Anger-out is defined as “expressing angry feelings in aggressive verbal or motor behavior directed toward other people or objects in the environment” (Spielberger et al., 1995, p. 57 and this book), and thus seems to encompass approach-motivated anger. In an important extension of this work, Rybak, Crayton, Young, Herba, and Konopka (2006) found that among adolescent male psychiatric patients, more symptoms of aggression and impulsivity related to greater relative left frontal activity.

Harmon-Jones (2004) addressed an alternative explanation for the above set of results. Hypothetically, persons with high levels of trait anger might experience anger as a positive emotion, and this positive attitude toward anger could be responsible for anger being associated with relative left frontal activity. After developing a valid and reliable assessment of attitude toward anger, we assessed whether resting baseline asymmetrical activity related to trait anger and/or attitude toward anger. Results indicated that relative left frontal activity related to anger, but not attitude toward anger (Harmon-Jones, 2004). Further partial correlational analyses revealed that the relationship between trait anger and left frontal activity was not mediated by an association between trait anger and having a positive attitude toward anger.

In an important complement to these results, Hewig et al. (2004) found that anger-control, as measured by the STAXI (Spielberger, 1988), was related to greater right than left frontal activity. Anger-control is considered as an active coping strategy to resolve or manage anger using nonaggressive behaviors or activities (Deffenbacher, Oetting, Lynch, & Morris, 1996; Schwenkmezger, Hodapp, & Spielberger, 1992). For instance, Deffenbacher et al. (1996) described anger-control as

“the tendency to engage in calming and palliative activities that lower arousal and calm the individual” (p. 576), and Schwenkmezger et al. (1992) considered anger-control as an active coping style to control the expression of anger or to express anger in a socially appropriate way. Hewig interpreted the association of anger-control with relative right activation as due to individuals high in anger-control typically responding with withdrawal motivation when angry.

5.4.6 Anger and EEG Activity – Experimental Studies of State Anger

To address the limitations of the above correlational studies, experiments have been conducted in which anger is manipulated and the corresponding effects on regional brain activity are examined. In Harmon-Jones and Sigelman (2001), participants were randomly assigned to a condition in which they were insulted or treated in a neutral manner by another ostensible participant. Immediately following the treatment, EEG data were collected. As predicted, individuals who were insulted showed greater relative left frontal activity than individuals who were not insulted. Additional analyses revealed that within the insult condition, self-reported anger and aggression were positively correlated with relative left frontal activity. Neither of these correlations was significant in the no-insult condition. These results suggest that relative left frontal activation was associated with the evocation of anger. This research provides the first demonstration of a relationship between state anger and relative left frontal activation.

Another experiment replicated these results and also revealed that state anger evokes both increased left and decreased right frontal activity. Moreover, a manipulation of sympathy for the person who would later insult the participant revealed that sympathy reduced the effects of insult on respective left and right frontal activity (Harmon-Jones, Vaughn-Scott, Mohr, Sigelman & Harmon-Jones, 2004). This research suggests that sympathy for another individual may reduce aggression toward that individual (e.g., see review by Miller & Eisenberg, 1988) by reducing the relative left frontal activity associated with anger.

In the two experiments just described, the designs were tailored to evoke approach-oriented anger. Although most instances of anger involve approach, as discussed earlier, not all are. To manipulate approach motivation independently of anger, Harmon-Jones, Sigelman, Bohlig and Harmon-Jones (2003) performed an experiment in which the ability to cope with the anger-producing event was manipulated. Based on past research that has revealed that coping potential affects motivational intensity (Brehm & Self, 1989), it was predicted that the expectation of being able to take action to resolve the anger-producing event would increase approach motivational intensity relative to expecting to be unable to take action.

To manipulate coping potential or the expectation that one can act to change the situation, the two conditions in this experiment differed with regard to whether it was possible for participants to change the event that caused the anger. Both conditions evoked significant increases in anger (over baseline) and were not significantly different from each other. More importantly, and consistent with predictions, participants who expected to engage in the approach-related action showed greater left frontal activity than participants who expected to be unable to engage in approach-related action. Moreover, within the action-possible condition, participants who showed greater left frontal activity in response to the angering event also showed greater self-reported anger. These results provided support for the idea that anger is often an approach-related emotional response. In the condition where action was not possible, greater left frontal activity did not relate to greater anger. In our view, this is because, although anger usually leads to approach motivation, when action is not possible, approach motivation may remain low, even if angry feelings are high. Finally, within the action-possible condition, participants who evidenced greater left frontal activity in response to the event

were more likely to engage in behaviors that would reduce the possibility of the angering event from occurring in the future (i.e., they were more likely to sign the petition and to take petitions with them for others to sign to prevent a possible tuition increase at their university). This finding suggests that greater approach motivation, as reflected in greater left frontal cortical activity, was associated with more action to correct the negative situation. This effect has recently been replicated (Harmon-Jones, Lueck, Fearn, & Harmon-Jones, 2006).

This research suggests that the left frontal region is most accurately described as a region sensitive to approach motivational intensity. It is only when anger is associated with an opportunity to behave in a manner to resolve the anger-producing event that participants show increased relative left frontal activation.

5.4.7 Trait × State Anger Interactions in Frontal Asymmetry

The results of these two experiments should not be taken to indicate that such explicit manipulations of action possibility are always necessary. Manipulations of action possibility may only potentiate the effects of emotion manipulations on asymmetrical frontal cortical activity. This is supported by the results of a study in which participants were passively exposed to anger-inducing pictures with no explicit action expectancy (positive, fear/disgust, and neutral pictures were also presented). Across all participants, there was no main effect of relative left frontal activation. However, individual differences in trait anger related to relative left frontal reaction to the anger-inducing pictures, such that individuals high in trait anger showed greater left frontal activity to the anger-producing pictures (controlling for activity to neutral pictures; Harmon-Jones, 2007, see Schultz, this book, for review of effects associated with high trait anger).

Jensen-Campbell, Knack, Waldrip, and Campbell (2007) examined the role of conscientiousness and agreeableness in influencing self-control during interpersonally frustrating situations. Conscientiousness is the trait of being painstaking and careful. It includes self-discipline, carefulness, thoroughness, organization, and deliberation (the tendency to think carefully before acting). Conscientiousness is related to emotional intelligence and impulse control. Agreeableness is the trait of being cooperative, empathetic, considerate, friendly, generous, helpful, and generally likable; the negative pole of this trait is unfriendliness and hostility. Conscientiousness, but not agreeableness, was inversely related to both self-reported anger and relative left frontal cortical activation following an interpersonal insult. These results suggest that individuals high in conscientiousness respond to interpersonal insults with lower anger-related left frontal activation, whereas individuals low in conscientiousness respond to the same insult with higher left frontal activation.

In another study, we (Harmon-Jones, Abramson, Sigelman, Bohlig, Hogan, & Harmon-Jones, 2002) tested the hypothesized link between hypomania/mania and increased reactivity of the BAS to anger-evoking stimuli (Depue & Iacono, 1989). Based on this hypothetical link, we predicted that proneness toward mania would be related to increased relative left frontal activity in response to an anger-evoking event. In contrast, we predicted that proneness toward unipolar depression would be related to decreased relative left frontal activity in response to the anger-evoking event. To assess these individual differences among a representative sample of undergraduate students, we used the General Behavior Inventory, which was developed to identify individuals who are at risk for developing these disorders (Depue & Klein, 1988; Depue, Krauss, Spont, & Arbisi, 1989). Results confirmed that tendencies toward mania related to increased left frontal activity, and that tendencies toward unipolar depression related to decreased left frontal activity. In these analyses, resting, baseline relative left frontal activity was statistically controlled, suggesting that the effects were specific to the arousal of anger.

5.4.8 Asymmetrical Frontal Activity and Withdrawal Anger

To separate motivational direction from affective valence, Wacker, Heldmann, and Stemmler (2003) conducted an experiment in which soccer players were instructed to imagine that they were unfairly prevented from playing a soccer game by the coach. In the anger-approach condition, the participants imagined approaching the coach and protesting, whereas in the anger-withdrawal condition, they imagined backing out of the locker room and swearing silently at the coach. Results revealed that both conditions evoked self-reported anger and relative left frontal activation. These results support the tight coupling of anger with approach motivation and illustrate the difficulty of evoking a withdrawal-oriented angry state.

To explore whether an angry-withdrawal motivating state would activate greater right than left frontal cortical activity, we designed a study to examine the joint influence of trait and situational factors that would engender a withdrawal-oriented expression of anger. Based on past research (Plant & Devine, 2003), we hypothesized that a potential interracial interaction may create withdrawal motivation because people are reluctant to express anger that would appear prejudicial. That is, anger in the context of an interracial interaction may prompt a desire to avoid the situation. EEG activity was measured while white participants anticipated an interaction with a black individual. Consistent with expectations, self-reported anger was associated with relative right frontal cortical activity. Moreover, anger was associated with greater anxiety and greater skin conductance levels. Seemingly, certain individuals in particular contexts may experience anger accompanied by withdrawal motivation.

5.4.9 Manipulating Asymmetrical Frontal Activity and Anger Processes

The studies discussed above provide evidence that anger is commonly associated with an increase in left frontal cortical activity, thus suggesting that such activity is associated with approach motivation rather than positive valence. If this association is causal, the converse follows: increases in left frontal cortical activity should be associated with increases in anger.

Asymmetrical frontal cortical activity can be initiated by contracting unilateral body muscles. For instance, past research has revealed that contractions of the right hand and of the right side of the face induce positive affect and assertiveness and bias perceptions and judgments positively (Schiff & Lamon, 1989, 1994). Other research has found that as compared to left-sided contractions, right-sided contractions caused greater persistence in attempting to solve insoluble problems (Schiff et al., 1998). One recent experiment revealed that right-hand contractions, as compared to left-hand contractions, cause increased left frontal cortical activity and increased PANAS PA to mildly positive approach stimuli (Harmon-Jones, 2006).

We hypothesized that increased left frontal cortical activity brought about by unilateral hand contractions would increase aggression following an insult. We recently conducted an experiment in which subjects made right vs. left-hand contractions for roughly 4 min. Then, they were given insulting feedback on an essay they had written earlier in the session. Following the insult, participants played a competitive reaction time game, ostensibly against the person who insulted them. Aggression was measured by allowing participants to choose the level and length of a blast of white noise to administer to the person who had insulted them if they were fastest to respond to the stimulus on the screen. The game was designed so approximately half of the trials were won and half were lost by the participant. As compared to participants who contracted their left hand, participants who contracted their right hand evidenced greater relative left cortical activation over central and frontal regions. EEG coherence analyses suggested that the motor strip activations caused by unilateral

hand contractions spread to the lateral frontal sites. Moreover, as compared to left-hand contraction participants, those who contracted their right hand delivered longer and louder noise blasts to their opponent, suggesting that they were more behaviorally aggressive (Peterson, Shackman, et al., 2008).

The most direct experimental evidence of the relationship between left frontal activation and anger was provided by d'Alfonso, van Honk, Hermans, Postma & de Haan (2000) who used slow repetitive transcranial magnetic stimulation (rTMS) to inhibit either left or right prefrontal cortex. Slow rTMS reduces cortical excitability, so that rTMS applied to the right prefrontal cortex decreases its activation and causes the left prefrontal cortex to become more active, whereas rTMS applied to the left prefrontal cortex causes activation of the right prefrontal cortex. They found that increased activation of the left prefrontal cortex caused participants to attentionally approach angry faces (as in an aggressive confrontation). In contrast, an increase in right prefrontal activity led participants to attentionally avoid angry faces (as in a fear-based avoidance). The interpretation of these results is supported by research demonstrating that attention toward angry faces is associated with high levels of self-reported anger, BAS, and testosterone, and that attention away from angry faces is associated with high levels of cortisol and social anxiety (Putman, Hermans, & van Honk, 2004; van Honk et al., 1998, 1999; van Honk, Tuiten, de Haan, van den Hout, Stam, 2001). Moreover, these effects have been conceptually replicated using a memory paradigm (van Honk & Schutter, 2006).

5.4.10 Other Brain-Imaging Methods/Alternative Interpretations

There have been several studies examining neural responses to photographs of angry faces. Because these studies are likely assessing neural processes associated with the perception of emotional stimuli and not necessarily the experience or expression of emotion, these studies are not reviewed.

The research reviewed here has revealed that the left frontal cortical region is involved in approach-motivated anger. Few studies using brain-imaging technologies other than EEG have been conducted. In one, positron emission tomography (PET²; oxygen-15-labeled carbon dioxide) was measured while men were exposed to personally created angry or neutral mental imagery scripts. Results revealed that as compared to neutral imagery, anger imagery caused an increase in the left orbital frontal and precentral cortices, the right anterior cingulate cortex, and bilaterally in medial frontal cortex, anterior temporal poles, and cerebellum. The increase in activity in the left orbital frontal cortex is consistent with the anger research results obtained using EEG. However, Dougherty et al. (1999) interpreted the increase in left orbital frontal cortical activity as corresponding “to inhibition of aggressive behavior in the face of anger” (p. 471). Whereas this interpretation is consistent with some speculations on the role of the left orbital frontal cortex in response inhibition (Mega, Cummings, Salloway, & Malloy, 1997), it is inconsistent with the EEG results showing that increased left frontal activity is associated with increased aggression and approach behavior (e.g., Harmon-Jones & Sigelman, 2001; Harmon-Jones, Harmon-Jones, Bohlig, & Harmon-Jones, 2003). The interpretation that the left frontal cortical region is involved in the inhibition of anger and aggression is also inconsistent with lesion data suggesting that mania results from damage to the right frontal region (e.g., Robinson & Downhill, 1995) and results obtained when the left relative to right frontal cortex is activated and angry attentional processes are measured (e.g., d'Alfonso et al., 2000). However, EEG is likely assessing dorsolateral frontal cortical activity and not orbital frontal activity (Pizzagalli, Sherwood, Henriques, & Davidson, 2005), and left orbital frontal activity may be involved in the inhibition of anger, whereas left dorsolateral frontal activity may be involved in the approach motivational processes associated with anger.

It may be difficult to compare the anger induced by imagery in the PET² experiments to the anger induced by insulting feedback or goal blocking in the EEG experiments. In the imagery experiments,

there was no report of a significant association between reported anger and regional brain activity. In the EEG experiments, self-reported anger has been found to correlate significantly with relative left frontal activity. Such correlations assist in determining whether the brain activation is related to emotional experience or some other nonemotional variable.

Some researchers have suggested that the left PFC region serves the function of down-regulating negative affect, and that high left frontal activation is a marker of psychological well-being (Davidson, Putnam, & Larson, 2000). However, the research on anger and asymmetrical frontal cortical activity, when considered in whole, strongly suggests that the relationship between frontal asymmetry and psychological well-being is more complex. That is, relative left frontal activation has been associated with self-reported state anger and behavioral aggression (Harmon-Jones & Sigelman, 2001) and approach-motivated behavior (Harmon-Jones et al., 2003). Individuals with proneness toward mania (Harmon-Jones et al., 2002) and individuals higher in trait anger (Harmon-Jones, 2007) show even greater relative left frontal activation in response to angering events. Moreover, manipulated increases in left frontal activation cause approach-related angry attentional, memory, and behavioral responses (d'Alfonso et al., 2000; Peterson, Shackman, & Harmon-Jones, 2008; van Honk & Schutter, 2006). Finally, even at resting baseline, individuals who are higher in trait anger show greater relative left frontal activity (Hewig et al., 2004; Harmon-Jones & Allen, 1998; Rybak et al., 2006), and this relationship also occurs in adolescents who are in psychiatric in-patient units for impulse control disorders (Harmon-Jones & Allen, 1998; Rybak et al., 2006). It would be illogical to suggest that all of these individuals are inhibiting anger more than individuals without high levels of state anger, trait anger, approach behavior, aggression, or mania, or to suggest that these individuals are uniformly higher in psychological well-being.

5.4.11 Some Questions and Conclusions

Of course, approach motivations such as anger involve a system of brain regions, but the reviewed research establishes the importance of the left prefrontal cortex in approach motivation independent of affective valence. Scientists often suggest that prefrontal cortex (PFC) is involved in higher level cognitive functions, such as working memory and inhibitory processes. Part of the reason scientists reserve the PFC for higher level cognitive processes is because it is much larger in humans than other animals. The logic continues that if the PFC were a relatively recent development in evolution, then it must be the source of those psychological processes that separate us from other animals. This logic is likely at least partially correct, but not foolproof. For example, recent single-cell research with rats has revealed that the PFC is involved in aggression and most of the cells activated are not inhibitory cells (Halász, Tóth, Kalló, Liposits, & Haller, 2006). The PFC is a vast territory and is likely involved in numerous psychological processes.

In addition, evidence suggests that various mental processes involve the activation of areas throughout the brain, rather than each process being localized in just one area. That is, different psychological functions may reside in very specific territories of the left PFC, and/or different neurons in the left PFC may be involved in the different psychological functions. At present, it is not clear which possibility is most likely. The size, complexity, and activity of the human PFC suggest that it is involved in many processes.

Humans are better able to plan behavior and control their responses to emotional stimuli than other animals. No doubt the PFC is involved in such planning and control. However, this planning and execution of behavior is not always in the service of inhibiting destructive motivations. In fact, some planned and effortful behaviors that are said to distinguish humans from other mammals, such as war and genocide, actually enhance the destructiveness of approach-oriented aggressive motivation.

Future research will need to explore connections between sub-cortical and cortical structures in approach and withdrawal motivation. Along these lines, some research suggests activations in the left frontal cortex are related to dopaminergic projections from the striatum associated with the coordination of action with learned reward contingencies (Berridge, Espana, & Stalnacher, 2003; Tomer, Goldstein, Wang, Wong, & Volkow, 2008). However, it is unlikely that the motivational-related activations observed in the frontal cortices are simply due to “propagation” of signals from solely sub-cortical structures, as source-localization analyses have suggested that approach-withdrawal-related frontal asymmetries reflect changes in dorsolateral prefrontal cortical activity (Pizzagalli et al., 2005).

The work on asymmetrical frontal cortical activity and anger has implications for the psychological theories of emotions and anger in particular. This research demonstrated that unlike other negative emotions, anger is often associated with approach motivational tendencies. Consequently, major dimensional theories of emotion will need to be modified to incorporate the idea that not all negative affects are associated with withdrawal motivation. Also, the research on anger suggested social situations and individual differences that may cause anger to be associated with withdrawal motivation. This work may have important implications for understanding the inhibition of aggressive behavior as well as the development and/or maintenance of anxiety disorders.

In conclusion, the bulk of the research suggests that greater left than right frontal cortical activity is associated with approach-motivated anger. More recent studies suggest, in contrast, that greater right than left frontal cortical activity is associated with withdrawal-motivated anger. The dynamic motivational properties of anger will be better understood if future research examines asymmetrical frontal cortical activations as they unfold in real time in more dynamic situations (Chapter 22 by M. Potegal, this volume).

Acknowledgments Portions of the research described within this chapter were supported by a grant from the National Science Foundation (BCS 0350435) and by a grant from the National Institute of Mental Health (R03 MH60747-01).

References

- Averill, J. (1982). *Anger and aggression: An essay on emotion*. New York: Springer-Vela.
- Baron, R. A. (1977). Effects of victim's pain cues, victim's race, and level of prior instigation upon physical aggression. *Journal of Applied Social Psychology*, 9, 103–114.
- Berkowitz, L. (1989). Frustration–aggression hypothesis: Examination and reformulation. *Psychological Bulletin*, 106, 59–73.
- Berkowitz, L. (1993). *Aggression: Its causes, consequences, and control*. New York: McGraw-Hill.
- Berkowitz, L. (1999). Anger. In T. Dalgleish & M. Power (Eds.), *Handbook of cognition and emotion* (pp. 411–428). Chichester: Wiley.
- Berkowitz, L., Cochran, S., & Embree, M. (1981). Physical pain and the goal of aversively stimulated aggression. *Journal of Personality and Social Psychology*, 40, 687–700.
- Berkowitz, L., & Harmon-Jones, E. (2004a). More thoughts about anger determinants. *Emotion*, 4, 151–155.
- Berkowitz, L., & Harmon-Jones, E. (2004b). Toward an understanding of the determinants of anger. *Emotion*, 4, 107–130.
- Berridge, C. W., Espana, R. A., & Stalnaker, T. A. (2003). Stress and coping: Asymmetry of dopamine efferents within prefrontal cortex. In K. Hugdahl & R. J. Davidson (Eds.), *The asymmetrical brain* (pp. 69–103). Cambridge, MA: MIT Press.
- Brehm, J. W., & Self, E. (1989). The intensity of motivation. In M. R. Rosenzweig & L. W. Porter (Eds.), *Annual review of psychology* (Vol. 40, pp. 109–131). Palo Alto, CA: Annual Reviews.
- Bush, G., Luu, P., & Posner, M. I. (2000). Cognitive and emotional influences in anterior cingulate cortex. *Trends in Cognitive Science*, 4, 215–222.
- Buss, A. H., & Perry, M. (1992). The aggression questionnaire. *Journal of Personality and Social Psychology*, 63, 452–459.

- Canli, T., Desmond, J. E., Zhao, Z., Glover, G., & Gabrieli, J. D. (1998). Hemispheric asymmetry for emotional stimuli detected with fMRI. *NeuroReport*, 9, 3233–3239.
- Carver, C. S. (2004). Negative affects deriving from the behavioral approach system. *Emotion*, 4, 3–22.
- Carver, C. S., & White, T. L. (1994). Behavioral inhibition, behavioral activation, and affective responses to impending reward and punishment: The BIS/BAS scales. *Journal of Personality and Social Psychology*, 67, 319–333.
- Cassidy, F., Forest, K., Murry, E., & Carroll, B. J. (1998). A factor analysis of the signs and symptoms of mania. *Archives of General Psychiatry*, 55, 27–32.
- Coan, J. A., & Allen, J. J. B. (2003). Frontal EEG asymmetry and the behavioral activation and inhibition systems. *Psychophysiology*, 40, 106–114.
- Cook, I. A., O'Hara, R., Uijtdehaage, S. H. J., Mandelkern, M., & Leuchter, A. F. (1998). Assessing the accuracy of topographic EEG mapping for determining local brain function. *Electroencephalography and Clinical Neurophysiology*, 107, 408–414.
- Cunningham, W. A., Espinet, S. D., DeYoung, C. G., & Zelazo, P. D. (2005). Attitudes to the right- and left: Frontal ERP asymmetries associated with stimulus valence and processing goals. *NeuroImage*, 28, 827–834.
- d'Alfonso, A. A. L., van Honk, J., Hermans, E., Postma, A., & de Haan, E. H. F. (2000). Laterality effects in selective attention to threat after repetitive transcranial magnetic stimulation at the prefrontal cortex in female subjects. *Neuroscience Letters*, 280, 195–198.
- Darwin, C. (1965). *The expression of the emotions in man and animals*. Chicago: University of Chicago Press Original work published 1872.
- Davidson, R. J. (1998). Anterior electrophysiological asymmetries, emotion and depression: Conceptual and methodological conundrums. *Psychophysiology*, 35, 607–614.
- Davidson, R. J., & Irwin, W. (1999). The functional neuroanatomy of emotion and affective style. *Trends in Cognitive Science*, 3, 11–21.
- Davidson, R. J., Putnam, K. M., & Larson, C. L. (2000). Dysfunction in the neural circuitry of emotion regulation—A possible prelude to violence. *Science*, 289, 591–594.
- Deffenbacher, J. L., Oetting, E. R., Lynch, R. S., & Morris, C. D. (1996). The expression of anger and its consequences. *Behaviour Research and Therapy*, 34, 575–590.
- Depue, R. A., & Iacono, W. G. (1989). Neurobehavioral aspects of affective disorders. *Annual Review of Psychology*, 40, 457–492.
- Depue, R. A., & Klein, D. N. (1988). Identification of unipolar and bipolar affective conditions in nonclinical and clinical populations by the General Behavior Inventory. In D. L. Dunner, E. S. Gershon, & J. E. Barrett (Eds.), *Relatives at risk for mental disorder* (pp. 179–202). New York: Raven Press.
- Depue, R. A., Krauss, S., Spoont, M. R., & Arbisi, P. (1989). General behavior inventory identification of unipolar and bipolar affective conditions in a nonclinical university population. *Journal of Abnormal Psychology*, 98, 117–126.
- Dollard, J., Doob, L., Miller, N., Mowrer, O., & Sears, R. (1939). *Frustration and aggression*. New Haven, CT: Yale University Press.
- Dougherty, D. D., Shin, L. M., Alpert, N. M., Pitman, R. K., Orr, S. P., Lasko, M., et al. (1999). Anger in health men: A PET study using script-driven imagery. *Biological Psychiatry*, 46, 466–472.
- Eckhardt, C. I., & Deffenbacher, J. L. (1995). Diagnosis of anger disorders. In H. Kassirer (Ed.), *Anger Disorders: Definition, diagnosis and treatment* (pp. 27–48). Boca Raton, FL: Taylor & Francis.
- Ekman, P., & Friesen, W. V. (1975). *Unmasking the face: A guide to recognizing emotions from facial clues*. Englewood Cliffs, NJ: Prentice-Hall.
- Ellsworth, P. C., & Scherer, K. R. (2003). Appraisal processes in emotion. In R. J. Davidson, H. Goldsmith, & K. R. Scherer (Eds.), *Handbook of the affective sciences* (pp. 572–595). New York: Oxford University Press.
- Fowles, D. C. (1993). Behavioral variables in psychopathology: A psychobiological perspective. In P. B. Sutker & H. E. Adams (Eds.), *Comprehensive handbook of psychopathology* (2nd ed., pp. 57–82). New York: Plenum.
- Fox, N. A., & Davidson, R. J. (1986). Taste-elicited changes in facial signs of emotion and the asymmetry of brain electrical activity in human newborns. *Neuropsychologia*, 24, 417–422.
- Frijda, N. H. (1986). *The emotions*. Cambridge: Cambridge University Press.
- Frijda, N. H. (1993). The place of appraisal in emotion. *Cognition and Emotion*, 7, 357–387.
- Frijda, N. H., Kuipers, P., & ter Schure, E. (1989). Relations among emotion, appraisal, and emotional action readiness. *Journal of Personality and Social Psychology*, 57, 212–228.
- Frijda, N. H., & Zeelenberg, M. (2001). Appraisal: What is the dependent? In K. R. Scherer, A. Schorr, & T. Johnstone (Eds.), *Appraisal processes in emotion* (pp. 141–155). Oxford: Oxford University Press.
- Goldman, R. I., Stern, J. M., Engel, J., Jr., & Cohen, M. S. (2002). Simultaneous EEG and fMRI of the alpha rhythm. *NeuroReport*, 13, 2487–2492.

- Gray, J. A. (1987). *The psychology of fear and stress*. London: Cambridge University Press.
- Halász, J., Tóth, M., Kalló, I., Liposits, Z., & Haller, J. (2006). The activation of prefrontal cortical neurons in aggression – A double labeling study. *Behavioural Brain Research*, 175, 166–175.
- Harmon-Jones, E. (2003). Anger and the behavioural approach system. *Personality and Individual Differences*, 35, 995–1005.
- Harmon-Jones, E. (2004). On the relationship of anterior brain activity and anger: Examining the role of attitude toward anger. *Cognition and Emotion*, 18, 337–361.
- Harmon-Jones, E. (2006). Unilateral right-hand contractions cause contralateral alpha power suppression and approach motivational affective experience. *Psychophysiology*, 43, 598–603.
- Harmon-Jones, E. (2007). Trait anger predicts relative left frontal cortical activation to anger-inducing stimuli. *International Journal of Psychophysiology*, 66, 154–160.
- Harmon-Jones, E., Abramson, L. Y., Sigelman, J., Bohlig, A., Hogan, M. E., & Harmon-Jones, C. (2002). Proneness to hypomania/mania or depression and asymmetrical frontal cortical responses to an anger-evoking event. *Journal of Personality and Social Psychology*, 82, 610–618.
- Harmon-Jones, E., & Allen, J. J. B. (1998). Anger and prefrontal brain activity: EEG asymmetry consistent with approach motivation despite negative affective valence. *Journal of Personality and Social Psychology*, 74, 1310–1316.
- Harmon-Jones, E., Harmon-Jones, C., Abramson, L., & Peterson, C. K. (2007). PANAS positive affect and anger. *Emotion*, 9, 183–196.
- Harmon-Jones, E., Lueck, L., Fearn, M., & Harmon-Jones, C. (2006). The effect of personal relevance and approach-related action expectation on relative left frontal cortical activity. *Psychological Science*, 17, 434–440.
- Harmon-Jones, E., & Sigelman, J. (2001). State anger and prefrontal brain activity: Evidence that insult-related relative left prefrontal activation is associated with experienced anger and aggression. *Journal of Personality and Social Psychology*, 80, 797–803.
- Harmon-Jones, E., Sigelman, J. D., Bohlig, A., & Harmon-Jones, C. (2003). Anger, coping, and frontal cortical activity: The effect of coping potential on anger-induced left frontal activity. *Cognition and Emotion*, 17, 1–24.
- Harmon-Jones, E., Vaughn-Scott, K., Mohr, S., Sigelman, J., & Harmon-Jones, C. (2004). The effect of manipulated sympathy and anger on left and right frontal cortical activity. *Emotion*, 4, 95–101.
- Hewig, J., Hagemann, D., Seifert, J., Naumann, E., & Bartussek, D. (2004). On the selective relation of frontal cortical asymmetry and anger-out versus anger-control. *Journal of Personality and Social Psychology*, 87, 926–939.
- Izard, C. E. (1977). *Human emotions*. New York: Plenum.
- Izard, C. E. (1991). *The psychology of emotions*. New York: Plenum.
- Jensen-Campbell, L. A., Knack, J. M., Waldrup, A. M., & Campbell, S. D. (2007). Do personality traits associated with self-control influence the regulation of anger and aggression? *Journal of Research in Personality*, 41, 403–424.
- Kalin, N. H., Shelton, S. E., Davidson, R. J., & Kelley, A. E. (2001). The primate amygdala mediates acute fear but not the behavioral and physiological components of anxious temperament. *Journal of Neuroscience*, 21, 2067–2074.
- Knutson, B., & Peterson, R. (2005). Neurally reconstructing expected utility. *Games and Economic Behavior*, 52, 305–315.
- Knutson, B., & Wimmer, G. E. (2007). Reward: Neural circuitry for social valuation. In E. Harmon-Jones & Winkelman, P. (Eds.), *Social neuroscience: Integrated biological, and psychological explanations of social behavior* (pp. 157–175). New York, NY: Guilford Press.
- Lang, P. J. (1995). The emotion probe: Studies of motivation and attention. *American Psychologist*, 50, 372–385.
- Lazarus, R. S. (1991). *Emotion and adaptation*. New York: Oxford University Press.
- Lerner, J. S., & Keltner, D. (2001). Fear, anger, and risk. *Journal of Personality and Social Psychology*, 81, 146–159.
- Lewis, M. (1993). The development of anger and rage. In R. A. Glick & S. P. Roose (Eds.), *Rage, power, and aggression* (pp. 148–168). New Haven, CT: Yale University Press.
- Lewis, M., Alessandri, S. M., & Sullivan, M. W. (1990). Violation of expectancy, loss of control, and anger expressions in young infants. *Developmental Psychology*, 26, 745–751.
- Lewis, M., Sullivan, M. W., Ramsey, D. S., & Alessandri, S. M. (1992). Individual differences in anger and sad expressions during extinction: Antecedents and consequences. *Infant Behavior and Development*, 15, 443–452.
- Lindsley, D. B., & Wicke, J. D. (1974). The electroencephalogram: Autonomous electrical activity in man and animals. In R. Thompson & M. N. Patterson (Eds.), *Bioelectric recording techniques* (pp. 3–79). New York: Academic Press.
- Malone, R. P., Delaney, M. A., Luebbert, J. F., Cater, J., & Campbell, M. (2000). A double-blind placebo-controlled study of lithium in hospitalized aggressive children and adolescents with conduct disorder. *Archives of General Psychiatry*, 57, 649–654.

- Mega, M. S., Cummings, J. L., Salloway, S., & Malloy, P. (1997). The limbic system: an anatomic, phylogenetic, and clinical perspective. *Journal of Neuropsychiatry and Clinical Neuroscience*, 9, 315–330.
- Mikulincer, M. (1988). Reactance and helplessness following exposure to unsolvable problems: The effects of attributional style. *Journal of Personality and Social Psychology*, 54, 679–686.
- Miller, P. A., & Eisenberg, N. (1988). The relation of empathy to aggressive and externalizing/antisocial behavior. *Psychological Bulletin*, 103, 324–344.
- Mowrer, O. H. (1960). *Learning theory and behavior*. New York: Wiley.
- Nunez, P. L., & Srinivasan, R. (2006). *Electric fields of the brain: The Neurophysics of EEG*. New York: Oxford University Press.
- Ortony, A., Clore, G. L., & Collins, A. (1988). *The cognitive structure of emotions*. New York: Cambridge University Press.
- Panksepp, J. (1994). The basics of basic emotions. In P. Ekman & R. J. Davidson (Eds.), *The nature of emotion: Fundamental questions* (pp. 20–24). New York: Oxford University Press.
- Panksepp, J. (1998). *Affective neuroscience: The foundations of human and animal emotions*. New York: Oxford University Press.
- Parkinson, B., & Manstead, A. S. R. (1992). Appraisal as a cause of emotion. In M. S. Clark (Ed.), *Review of personality and social psychology* (Vol. 13, pp. 122–149). Newbury Park, CA: Sage.
- Peterson, C. K., Gable, P., & Harmon-Jones, E. (in press). Asymmetrical frontal ERPs, emotion, and behavioral approach/inhibition sensitivity. *Social Neuroscience*, 3, 113–124.
- Peterson, C. K., Shackman, A. J., & Harmon-Jones, E. (in press). The role of asymmetrical frontal cortical activity in aggression. *Psychophysiology*, 45, 86–92.
- Pizzagalli, D. A., Sherwood, R. J., Henriques, J. B., & Davidson, R. J. (2005). Frontal brain asymmetry and reward responsiveness. A source-localization study. *Psychological Science*, 16, 805–813.
- Plant, E. A., & Devine, P. G. (2003). The antecedents and implications of interracial anxiety. *Personality and Social Psychology Bulletin*, 29, 790–801.
- Plutchik, R. (1980). *Emotion: A psychoevolutionary synthesis*. New York: Harper & Row.
- Putman, P., Hermans, E., & van Honk, J. (2004). Emotional stroop performance for masked angry faces: It's BAS, not BIS. *Emotion*, 4, 305–311.
- Robinson, R. G., & Downhill, J. E. (1995). Lateralization of psychopathology in response to focal brain injury. In R. J. Davidson & K. Hugdahl (Eds.), *Brain asymmetry* (pp. 693–711). Cambridge: MIT Press.
- Robinson, R. G., Kubos, K. L., Starr, L. B., Rao, K., & Price, T. R. (1984). Mood disorders in stroke patients: Importance of location of lesion. *Brain*, 107, 81–93.
- Rolls, E. T. (1999). *The brain and emotion*. Oxford: Oxford University Press.
- Roseman, I. J. (1991). Appraisal determinants of discrete emotions. *Cognition and Emotion*, 5, 161–200.
- Roseman, I. J., Spindel, M. S., & Jose, P. E. (1990). Appraisals of emotion-eliciting events: Testing a theory of discrete emotions. *Journal of Personality and Social Psychology*, 59, 899–915.
- Russell, J. A., & Fernández-Dols, J. M. (1997). *The psychology of facial expression*. Paris: Cambridge University Press.
- Rybak, M., Crayton, J. W., Young, I. J., Herba, E., & Konopka, L. M. (2006). Frontal alpha power asymmetry in aggressive children and adolescents with mood and disruptive behavior disorders. *Clinical EEG and Neuroscience*, 37, 16–24.
- Scherer, K. R. (2001). Appraisal considered as a process of multilevel sequential checking. In K. R. Scherer, A. Schorr, & T. Johnstone (Eds.), *Appraisal processes in emotion* (pp. 92–120). Oxford: Oxford University Press.
- Schiff, B. B., & Bassel, C. (1996). Effects of asymmetrical hemispheric activation on approach and withdrawal responses. *Neuropsychology*, 10, 557–564.
- Schiff, B. B., Guirguis, M., Kenwood, C., & Herman, C. P. (1998). Asymmetrical hemispheric activation and behavioral persistence: Effects of unilateral muscle contractions. *Neuropsychology*, 12, 526–532.
- Schiff, B. B., & Lamon, M. (1989). Inducing emotion by unilateral contraction of facial muscles: a new look at hemispheric specialization and the experience of emotion. *Neuropsychologia*, 27, 923–935.
- Schiff, B. B., & Lamon, M. (1994). Inducing emotion by unilateral contraction of hand muscles. *Cortex*, 30, 247–254.
- Schwenkmezger, P., Hodapp, V., & Spielberger, C. D. (1992). *Das State-Trait-Aergerausdrucks-Inventar STAXI [State-Trait Anger Expression Inventory]*. Bern: Verlag Hans Huber.
- Shaver, P., Schwartz, J., Kirson, D., & O'Connor, C. (1987). Emotion knowledge: Further exploration of a prototype approach. *Journal of Personality and Social Psychology*, 52, 1061–1086.
- Silberman, E. K., & Weingartner, H. (1986). Hemispheric lateralization of functions related to emotion. *Brain & Cognition*, 5, 322–353.

- Sobotka, S. S., Davidson, R. J., & Senulis, J. (1992). Anterior brain electrical asymmetries in response to reward and punishment. *Electroencephalography and Clinical Neurophysiology*, 83, 236–247.
- Spielberger, C. D. (1988). *Professional manual for the state-trait anger expression inventory (STAXI)* (Research ed.). Odessa, FL: Psychological Assessment Resources.
- Spielberger, C. D., Jacobs, G. A., Russell, S. F., & Crane, R. S. (1983). Assessment of anger: The State-Trait Anger scale. In J. N. Butcher & C. D. Spielberger (Eds.), *Advances in personality assessment* (Vol. 2, pp. 159–187). Hillsdale, NJ: Lawrence Erlbaum Associates.
- Spielberger, C. D., Reheiser, E. C., & Sydeman, S. J. (1995). Measuring the experience, expression, and control of anger. In H. Kassirnové (Ed.), *Anger disorders: Definition, diagnosis, and treatment* (pp. 49–67). Washington, DC: Taylor & Francis.
- Stein, N. L., & Levine, L. J. (1989). The causal organization of emotional knowledge: A developmental study. *Cognition and Emotion*, 3, 343–378.
- Thut, G., Schultz, W., Roelcke, U., Nienhusmeier, M., Missimer, J., Maguire, R. P., et al. (1997). Activation of the human brain by monetary reward. *NeuroReport*, 8, 1225–1228.
- Tomer, R., Goldstein, R. Z., Wang, G. J., Wong, C., & Volkow, N. D. (in press). Incentive motivation is associated with striatal dopamine asymmetry. *Biological Psychology*, 77, 98–101.
- Tyrer, S., & Shopsin, B. (1982). Symptoms and assessment of mania. In E. S. Paykel (Ed.), *Handbook of affective disorders* (pp. 12–23). New York: Guilford.
- Vallortigara, G., & Rogers, L. J. (2005). Survival with an asymmetrical brain: Advantages and disadvantages of cerebral lateralization. *Behavioral and Brain Sciences*, 28, 575–589.
- van Honk, J., & Schutter, D. J. L. G. (2006). From affective valence to motivational direction. *Psychological Science*, 17, 963–965.
- van Honk, J., Schutter, D. J. L. G., d'Alfonso, A. A. L., Kessels, R. P. C., & de Haan, E. H. F. (2002). 1 hz rTMS over the right prefrontal cortex reduces vigilant attention to unmasked but not to masked fearful faces. *Biological Psychiatry*, 52, 312–317.
- van Honk, J., Tuiten, A., de Haan, E., van den Hout, M., & Stam, H. (2001). Attentional biases for angry faces: Relationships to trait anger and anxiety. *Cognition and Emotion*, 15, 279–297.
- van Honk, J., Tuiten, A., van den Hout, M., Koppeschaar, H., Thijssen, J., de Haan, E., et al. (1998). Baseline salivary cortisol levels and preconscious selective attention for threat: A pilot study. *Psychoneuroendocrinology*, 23, 741–747.
- van Honk, J., Tuiten, A., Verbaten, R., van den Hout, M., Koppeschaar, H., Thijssen, J., et al. (1999). Correlations among salivary testosterone, mood, and selective attention to threat in humans. *Hormones and Behavior*, 36, 17–24.
- Wacker, J., Heldmann, M., & Stemmler, G. (2003). Separating emotion and motivational direction in fear and anger: effects on frontal asymmetry. *Emotion*, 3, 167–193.
- Watson, D. (2000). *Mood and temperament*. New York: Guilford.
- Watson, D., & Clark, L. A. (1991). *The PANAS-X: Preliminary manual for the positive and negative affect schedule-expanded form*. Unpublished manuscript.
- Watson, D., Clark, L. A., & Tellegen, A. (1988). Development and validation of brief measures of positive and negative affect: The PANAS scales. *Journal of Personality and Social Psychology*, 54, 1063–1070.
- Wortman, C. B., & Brehm, J. W. (1975). Responses to uncontrollable outcomes: An integration of reactance theory and the learned helplessness model. In L. Berkowitz (Ed.), *Advances in experimental social psychology* (Vol. 8, pp. 278–336). New York: Academic Press.
- Young, P. T. (1943). *Emotion in man and animal: Its nature and relation to attitude and motive*. New York: Wiley.

Chapter 6

The Neurochemistry and Psychopharmacology of Anger

Alyson J. Bond and Janet Wingrove

Abstract Work investigating the relationship between neurotransmitter function and anger currently relies on indirect measures, such as levels of metabolites in cerebrospinal fluid, neuroendocrine challenges and monoamine depletion. The evidence from all three types of neurochemical study has supported deficient serotonin as the neurotransmitter most involved in angry aggression and to a lesser extent in the experience of anger itself. Experimental findings also demonstrate that a well-functioning 5-HT system is involved in anger regulation. Psychopharmacology studies support the experimental neurochemical work indicating the importance of individual differences in trait aggression, irrespective of diagnosis, in the experience and display of anger. Serotonergic antidepressants have been shown not only to have positive effects on reducing anger-related emotions but also to increase affiliative or cooperative behaviour. Mood stabilisers and antipsychotics with effects on 5-HT reduce anger and irritability in various patient groups. Selective noradrenergic antidepressants improve negative mood and can also exert pro-social effects but effects on anger per se have not been demonstrated.

6.1 Introduction

The neurochemistry and psychopharmacology of anger is a neglected field. Work has focused on aggressive behaviour because of the consequences. For example a study by Moller et al. (1996) used the Kinsey Institute Reaction II which asks subjects “How irritated or angry/upset do you become?” in particular situations. However, the intensity of the aggressive reaction was rated and not the degree of the emotion or affect felt. Anger is a negative emotion which is commonly experienced and not generally treated. However, anger is highly correlated with other negative emotions such as irritability, attitudes such as hostility and behaviours such as verbal and physical aggression (Archer, 2006) and impulsivity or loss of control (Baumeister, 1994) and therefore in some cases can be destructive.

Aggression has been classified into two essential types in both human and preclinical work: instrumental or predatory and affective or defensive (cf. Baron, 1977). “Instrumental” aggression is related to behaviour where the main goal is to establish social and coercive power over others and where aggressive behaviour has been judged as the most likely means to achieve this end.

A.J. Bond (✉)
Institute of Psychiatry, King's College London, London, UK
e-mail: a.bond@iop.kcl.ac.uk

“Affective” aggression is accompanied by a strong negative emotional state which is usually, but not always, labelled as anger in humans. It is accompanied by intense autonomic arousal (Chapter 7 by G. Stemmler, this book) and has been shown to be associated with decreased serotonergic and increased noradrenergic and dopaminergic activity in preclinical studies (Coccaro & Kavoussi, 1996). These two types are not as easy to differentiate in humans as they fail to capture the multiple motives often associated with aggressive acts (Bushman & Anderson, 2001). It has also been argued that both affective and instrumental aggression are directed towards the attainment of specific goals and are therefore both instrumental (Bandura, 1973). In response to these difficulties, various terms have been coined in association with different theories, e.g. proactive and reactive (Dodge & Coie, 1987). However, impulsive aggression and violence have been claimed to arise as a consequence of faulty emotion regulation (Davidson, Jackson, & Kalin, 2000) and difficulty with anger regulation is a feature of several psychiatric disorders (Chapter 27 by R.W. Novaco, this book). The study of the neurochemistry of anger can give us some indications of how to treat it pharmacologically if necessary.

6.2 Neurochemistry of Anger

Work investigating the relationship between various measures or indicators of neurotransmitter function and negative aspects of emotion or behaviour has generally focused on aggression rather than anger. This research is based on the assumption, for which there is considerable empirical support (e.g. Olweus, 1979), that aggression is a reasonably enduring or trait-like characteristic of individuals. Therefore the research has looked for associations between individual differences in aggression and measures of serotonergic function. However, people high in trait aggression are also likely to be high in trait anger and to respond to threatening, aversive or provocative events with higher rates of anger and hostility (Bond, Ruaro, & Wingrove, 2006).

Research into neurobiological mechanisms behind human aggression requires valid, reliable research tools to measure aggression and anger. Although commonly used in clinical trials, observer scales like the Overt Aggression Scale (OAS; Yudofsky, Silver, Jackson, Endicott, & Williams, 1986) measure only obvious acts of aggression. However, self-rated questionnaires measure different aspects of aggression including anger and can be used in many populations. The most commonly used questionnaire has been the Buss–Durkee Hostility Inventory (BDHI; Buss & Durkee, 1957) and this measures irritability, which can be understood as an emotional component of anger, as well as aggression (Table 6.1). The BDHI has since been refined into the Aggression Questionnaire (Buss & Perry, 1992) which has a measure of anger.

Because of the difficulties in assessing central neurotransmitter activity directly in humans, studies need to rely on indirect measures, such as levels of metabolites in cerebrospinal fluid (CSF), neuroendocrine challenges and monoamine depletion. Thus to assess central serotonergic activity, the levels of the metabolite of serotonin, 5-hydroxyindoleacetic acid (5-HIAA), have been measured, and to assess central noradrenergic activity, the levels of the metabolite of noradrenaline, 3-methoxy-4-hydroxyphenyl-glycol (MHPG), have been measured in cerebrospinal fluid (CSF) and correlated with measures of aggression and irritability. In neuroendocrine challenges, a drug with a specific action on a particular neurotransmitter is administered orally or sometimes intravenously, and the resulting change in hormone levels is correlated with individual differences in measures of aggression and anger. Finally, monoamine depletion is a way of temporarily lowering the functioning of central monoamine systems and responses to these manipulations can be measured. The first two methods have concentrated on associations with trait measures but the third assesses emotion

Table 6.1 Commonly used measures of anger, irritability and hostility in neurobiological studies

Buss–Durkee Hostility Inventory	Aggression Questionnaire
Assault	Physical aggression
Verbal hostility	Verbal aggression
Irritability	Anger
Indirect hostility	Hostility
Resentment	
Suspicion	
Guilt ^a	
Total score	Total score

^aNot included in total score.

change, although both trait and state measures have been used, e.g. to assess change in anger in people with different levels of trait aggression. Only studies, which have direct relevance for anger, will be reviewed.

6.2.1 Studies Investigating Metabolites of Serotonin in Cerebrospinal Fluid

Several research groups have investigated possible associations between CSF levels of 5-HIAA, the breakdown product of serotonin, and psychological characteristics, aspects of behaviour or psychiatric disorders. The most robust finding to emerge from this research is an association between low levels of CSF 5-HIAA and a history of suicide attempts (Lester, 1995; Mann, Waternaux, Haas, & Malone, 1999). This is clearly encouraging for the investigation of other characteristics, such as aggression, in demonstrating that meaningful relationships can be found and supporting the assumption that CSF 5-HIAA does reflect aspects of central serotonergic function. However, several studies investigating the relationship between CSF 5-HIAA have been confounded by failing to exclude participants with a history of suicide attempts. To elucidate this issue, Stanley et al. (2000) carried out a well-controlled study in psychiatric patients with a variety of Axis I disorders, excluding anyone with a history of suicide attempts. Using a median split on the Life History of Aggression (LHA), they found lower CSF 5-HIAA values in the high aggression group, confirming previous research. Virkkunen, Nuutila, Goodwin, and Linnoila (1987) also found that their group of violent offenders still had lower CSF 5-HIAA values than the healthy controls, when those with suicide attempts were excluded from the analysis. In addition, Placidi et al. (2001), using regression analysis, found that LHA was a better predictor of CSF 5-HIAA than history of suicide attempts or than severity of depression. These studies have all concentrated on measures of aggression but similar relationships have been shown with the irritability scale of the BDHI (Brown & Goodwin, 1984); CSF 5-HIAA showed a significant inverse correlation with irritability.

Poor functioning of the serotonergic system, demonstrated in lower CSF 5-HIAA values, has been associated with impulsive, hostile aggressive behaviour rather than instrumental, premeditated aggression (Linnoila et al., 1983; Virkkunen et al., 1987). This indicates that anger is an important constituent but it has rarely been examined separately. In a review covering much of the work, Tuinier, Verhoeven, and van Praag (1995) concluded that, taking methodological limitations into account, the evidence pointed in the direction of an association between low CSF 5-HIAA and outward aggression. However, this had only been established “for white male patients, who in addition are relatively young, personality disordered and have a history of criminal acts” (p. 154). There is still little evidence as to whether any relationship that exists in patient/offender groups extends into the general population.

In a study of healthy volunteers, Roy, Adinoff, and Linnoila (1988) found a correlation between CSF 5-HIAA and hostility in healthy volunteers in the expected direction, but this was not significant after controlling for age. The sample was possibly too small and heterogeneous for much to be concluded. On the other hand, Moller et al. (1996) used a considerably larger sample and found a significant correlation, but in the opposite direction; high scores on a questionnaire measure of other-directed aggression were associated with high CSF 5-HIAA levels. A recent study in depressed patients (Prochazka & Agren, 2003) also found a positive relationship between hostility on the AQ and CSF 5-HIAA only in males. This study was one of the few to also measure CSF MHPG and a positive relationship between CSF MHPG and hostility was found in the total sample and separately in females. These results confirmed the results of an earlier study (Roy, De Jong, & Linnoila, 1989). These authors also found that CSF concentrations of MHPG correlated positively with irritability and impulsivity, indicating that irritability is associated with enhanced noradrenergic activity.

The current evidence indicates that it is possibly only severe affective aggression, i.e. with accompanying anger, that is associated with low CSF 5-HIAA. However, there is as yet insufficient evidence from healthy volunteers to draw any firm conclusions. As obtaining a CSF sample via a lumbar puncture is an intrusive procedure which, for ethical reasons, is not approved for non-clinical indications in many countries, such work seems unlikely to be completed and the emphasis has thus shifted to other techniques.

6.2.2 Neuroendocrine Challenge Studies

In a neuroendocrine challenge the responsiveness of some part of the neurotransmitter system is assessed by measuring the change (usually an increase) in hormone release following acute administration of a single dose of a centrally acting agent. The hormones that have most commonly been measured are prolactin, growth hormone and cortisol.

6.2.3 The 5-HT System

Fenfluramine, a centrally active 5-HT releasing agent with reuptake inhibition properties, and its *d*-isomer have been used as a challenge agent to measure serotonergic activity in many different disorders (Newman, Shapira, & Lerer, 1998). Prolactin (PRL) responses to fenfluramine have been found to be blunted in patients with personality and mood disorders but only those with personality disorders showed inverse correlations with measures of aggression and impulsivity (Coccaro, Siever, Owen, & Davis, 1989). Anger is a component of many personality disorders but initially inverse correlations were only found with behavioural measures. A larger, more recently reported study by New et al. (2004) found a significant negative correlation between the PRL response to fenfluramine and a composite of the irritability and assault subscales of the Buss–Durkee Hostility Inventory (BDHI: Buss & Durkee, 1957) in a combined patient and healthy volunteer group of men, and in a subgroup of 29 men with intermittent explosive disorder. However, the authors did not separate the emotional (irritability) from the behavioural component. Fava et al. (2000) studied 22 men and 15 women with depression and found lower PRL response to fenfluramine in those with anger attacks. The difference remained significant after controlling for sex but again it is not known if serotonergic dysfunction was related only to the behavioural expression of anger.

Some studies have been carried out in healthy volunteers but these too have concentrated on behavioural rather than emotional indices. Cleare and Bond (1997) found no significant correlations between scores on the BDHI and the PRL response to 30 mg *d*-fenfluramine in either male or female

healthy volunteers. However, they did find a significant negative correlation between BDHI scores and the cortisol response to d-fenfluramine in the male participants, providing some support for the view that serotonergic function is related to hostility in the wider male population. The irritability scale alone did not show a significant correlation. The relationship between the cortisol response and anger, measured by the Spielberger Trait Anger Scale (STAS; Spielberger, Jacobs, Russell, & Crane, 1983), was in the same direction but failed to reach significance. Manuck et al. (1998) carried out a fenfluramine challenge (30–60 mg dose adjusted for body weight) in a large community sample. They found inverse correlations between peak plasma levels of PRL and measures of irritability and angry hostility as well as aggression in males. In addition, they found positive correlations with conscientiousness in both male and postmenopausal female volunteers. Flory, Manuck, Matthews and Muldoon (2004) examined the relationship between the PRL response to fenfluramine and daily ratings of positive and negative mood in a healthy sample. They found that the peak PRL effect was correlated with positive mood averaged over 7 days, but not with negative mood. This relationship between a normally functioning 5-HT system and positive traits is interesting, as most research has focused on the association between deficiencies in central serotonergic function and negative affects and traits. It supports a role for a well-functioning serotonin system in effective mood regulation. A second study (Manuck, Flory, Muldoon, & Ferrell, 2002) confirmed the relationship between PRL responses to fenfluramine and aggression in men. In order to be certain that the relationship was not caused by a few extreme scores that might be due to a few participants with unnoticed personality disorders in the sample, they demonstrated that the correlation was still evident when they excluded the top and bottom quintiles from the distribution. Thus overall the evidence suggests an association between aggression/impulsivity and a blunted prolactin response to fenfluramine in the general population, in men at least, with lower scores in those with borderline or antisocial personality disorders.

Challenges have also been carried out using meta-chlorophenylpiperazine (m-cpp), a 5-HT agonist, which leads to reliable increases in ACTH, cortisol and prolactin, in a dose-dependent manner (Kahn & Wetzler, 1991). Based on evidence of an association between aggression and low levels of serotonin, Wetzler, Kahn, Asnis, Korn, and van Praag (1991) used a low dose of m-cpp to test the hypothesis of enhanced post-synaptic receptor sensitivity in aggression in a group of male and female patients with Axis I disorders and healthy volunteers. However, no differences in the PRL response to m-cpp were found between those with and without anger problems. Furthermore, there were no significant correlations with the BDHI. This negative result could be due to the low dose used in this study. Stein et al. (1996) also found no significant correlations between the PRL response to m-cpp and measures of aggression and impulsivity, including the BDHI, in either healthy controls or patients with borderline personality disorder but they did not specifically examine anger. Klaassen, Riedel, van Praag, Menheere, and Griez (2004) found no relationship between PRL response to m-cpp and trait anger in either healthy volunteers or patients with depressed mood (some with anxiety disorders and some with depression). They did find a positive correlation between the cortisol response to m-cpp and trait anger in the depressed mood group, but considered this to be a statistical artefact. Paris et al. (2004) studied women with borderline personality disorder and healthy controls and found a negative correlation between the PRL response to m-cpp and the BDHI in the total group and also in the BPD group alone (but not the healthy controls alone) but they did not discriminate between the irritability and aggression scales. Rinne, Westenberg, den Boer, & van den Brink (2000) found a lower PRL response to m-cpp in women with borderline personality disorder than in controls and also, within the BPD group, a negative correlation between the prolactin response to m-cpp and the frequency and duration of childhood abuse. Therefore there is relatively little evidence to suggest a link between responses to m-cpp and anger which may be partly due to its very variable pharmacokinetics (Gijsman et al., 1998).

Challenges have also been carried out using the partial 5-HT_{1A} agonists, buspirone and ipsapirone. A blunted PRL response to buspirone has been shown (Coccaro, Gabriel, & Siever, 1990) and in this study, an inverse relationship with self-reported irritability was found. The cortisol response to ipsapirone has also been found to correlate inversely not only with measures of trait and state aggression but also with angry feelings in a group of healthy volunteers who scored high on trait aggression measured by the BDHI (Cleare & Bond, 2000). However, Klaassen et al. (2004) found no significant correlations between trait anger and the cortisol, ACTH or prolactin responses to ipsapirone in patients with depressed mood or in healthy controls.

Thus, overall the neuroendocrine challenge studies using fenfluramine provide some evidence for an association between aggression and lower levels of serotonergic function, supporting the findings from the CSF 5-HIAA studies. In addition, the work with buspirone and ipsapirone supports this relationship. Although this work has largely concentrated on trait measures of aggression, hypo-functioning of the serotonergic system has also been associated with raised levels of irritability and anger. As serotonin is an inhibitory neurotransmitter, this could be understood as the low levels exhibited in personality disordered patients indicating difficulties in inhibiting aggressive responses to aversive stimuli. In less disordered patients or volunteers, any deficiency might only be shown as anger or irritability.

6.2.4 The Noradrenergic System

Clonidine, an alpha-2-adrenergic receptor agonist which stimulates growth hormone release, is the drug which has been used most often as a challenge agent to measure noradrenergic activity in different disorders. Growth hormone (GH) responses to clonidine have generally been found to be blunted in patients with depression (Van Moffaert & Dierick, 1999). GH responses to clonidine have also been found to be blunted in depressed patients compared to healthy controls and to improve after treatment with ECT or desipramine, a noradrenergic antidepressant (Coote, Wilkins, Werstiuk, & Steiner, 1998). Other noradrenergic challenges have shown similar results in depressed patients. Blunted GH responses to desipramine challenge and blunted cortisol responses to a metamphetamine challenge have been found (Siever, Coccaro, & Davis, 1986). Anger or irritability has not been measured in these studies.

Responses to clonidine have also been found to be blunted in other disorders in which anger is prominent such as post-traumatic stress disorder (PTSD). For example, Marshall et al. (2002) found patients with PTSD had lower cortisol, lower MHPG and reduced MHPG volatility to clonidine challenge than patients with panic disorder or healthy controls. Coccaro et al. (1991) explored the relationship between noradrenergic receptor responsivity and irritability and aggression. They measured GH responses to infusions with clonidine in healthy volunteers and patients with affective or personality disorders. GH values were found to correlate significantly positively with irritability but not aggression in the whole sample. Trestman et al. (1992) followed up this result in a larger overlapping study and confirmed a positive correlation between GH responses to clonidine and irritability and verbal aggression on the BDHI in patients with affective and personality disorders.

The limited evidence available from neuroendocrine studies using clonidine suggests some disparate results. Reduced responses to clonidine were found in patients with PTSD but hyperfunctioning of the noradrenergic system was associated with irritability in a mixed group of patients with affective or personality disorders. This suggests that drug treatments, which stabilise or diminish noradrenergic activity, might improve symptoms of irritability and anger.

6.2.5 Acute Monoamine Depletion

Certain experimental techniques have been used to temporarily lower the functioning of central monoamine systems: acute tryptophan depletion (TD), acute phenylalanine/tyrosine depletion (APTD), alpha-methyl-para-tyrosine (AMPT) and parachlorophenylalanine (PCPA). These manipulations have been shown to be specific to one or two neurotransmitter systems, specifically serotonin (TD; PCPA) or noradrenaline and dopamine (APTD; AMPT), and so represent a useful indirect way of studying neurochemical changes. However, most of the work has focused on depression (Booji, Van der Does, & Riedel, 2003). The lowering of all three of these neurotransmitters might be expected to increase negative affect but for the purposes of this chapter, only work, which has examined changes in negative mood related to anger, will be reviewed. AMPT is an inhibitor of tyrosine hydroxylase which impairs the synthesis of catecholamines, affecting both the dopamine and the noradrenaline systems. It has been used to study depressed patients and has been shown to induce temporary relapse in remitted patients and those being treated with noradrenergic medications (Booij et al., 2003). However, it can cause acute dystonic reactions and there is little evidence of mood effects in healthy volunteers and no evidence of effects on anger and so it will not be discussed further. Parachlorophenylalanine (PCPA) is a selective inhibitor of tryptophan hydroxylase and therefore lowers central serotonergic function but its use has been limited to preclinical work due its toxicity.

6.2.6 Acute Tryptophan Deletion

Acute tryptophan depletion is a method of acutely lowering levels of central serotonin by reducing the availability of its precursor, tryptophan. A drink containing 15 amino acids including the large neutral amino acids (LNAAs) but excluding tryptophan (Young, Smith, Pihl & Ervin, 1985) is administered. This drink has been shown to substantially lower both tryptophan in the plasma (up to 90%) and tissues and the rate of serotonin synthesis in the brain (Nishizawa et al., 1997) and the effects have been found to be maximal 4–6 h after consumption. The depleted drink is usually compared to either a balanced drink, with a similar composition but containing 2.3 g tryptophan, or an enhanced drink again with a similar composition but containing 10.3 g tryptophan. This technique has been used in many studies investigating depressed mood, in some studies of aggressive behaviour and a few have looked at more general mood effects. This section will focus on studies examining negative mood related to anger, hostility or irritability.

Danjou et al. (1990) compared two groups of nine male volunteers administered a 100 mg tryptophan depleted (TD) or a balanced drink on 33 visual analogue scales based on items from the Multiple Affect Adjective Checklist (MAACL: Zuckerman and Lubin, 1975). They found a significant effect of TD on one item only; “angry” which increased in the depleted group. Some studies have set out specifically to examine the effects of tryptophan manipulations on self-reports of angry or hostile mood. In one study, men selected to score high on the BDHI were randomly assigned to either 100 g of TD or enhancement (TE) (Cleare & Bond, 1994). It was found that the manipulations produced opposite and equal effects on a state measure of anger, the Anger Rating Scale (ARS: Bond and Lader, 1986). The TD group rated themselves higher, and the TE group lower, on anger. This supports the positive effects of enhancing serotonin on mood which may be particularly apparent in those with naturally deficient levels. In a second study (Cleare & Bond, 1995) the high hostility group was compared to a low hostility group. These two groups could also be differentiated on trait anger (STAS). No effects were shown in subjects selected to score low on the BDHI or STAS,

who presumably had well-functioning serotonergic systems, but the high hostility/anger group rated increased negative emotion on measures of both anger (ARS) and discontent on the Mood Rating Scale (MRS; Bond & Lader, 1974) when administered TD compared to TE. Thus the high and low hostility groups differed in their responses to the tryptophan manipulations, revealing that those with a predisposition to hostility were more sensitive to the effects. This finding was confirmed by Finn, Young, Pihl, and Ervin (1998). Different degrees of TD (100, 75, 50 and 25 g) or a balanced drink were administered to independent groups of subjects and changes in mood were measured with the MAACL. Change in plasma tryptophan was found to be significantly negatively associated with change in hostile mood. When subjects were divided into groups with high and low trait hostility according to the Cook–Medley Hostility Scale (Cook & Medley, 1954), there was a significant difference between the correlations for the two groups. The relationship between depletion and increased hostile mood was much stronger and only reached significance in the high trait subjects. The authors comment that although they did not use a TE condition, “much of the tryptophan-induced mood change was due to the decrease in hostility in persons whose plasma tryptophan increased after the balanced drink”. Wingrove, Bond, and Cleare (1999) compared TD and TE in high trait hostility men. Although depletion caused a similar increase in anger on the ARS as in previous studies, this was not significant due to an increase that also occurred following enhancement. However, subjects on TD did rate themselves as significantly more restless than those on TE and significant correlations were found between scores on the assault scale of the BDHI and increased anger after a laboratory task in the TD group. This result again demonstrates the vulnerability of those with an aggressive disposition to the mood effects of TD.

One study has examined the effects of TD on anger in psychiatric patients (Salomon, Mazure, Delgado, Mendia, & Charney, 1994). The effects of TD were compared to a balanced drink in a group of patients with intermittent explosive disorder. No significant effects were observed on the OAS or a “state-sensitive” version of the BDHI. This may be because these measures are related to behaviour and disposition rather than mood. Interestingly, irritability, the subscale most related to angry mood, did lessen significantly on the day after TD compared to the control drink. This suggests that the patients were aware when subtle mood changes, caused by depletion, resolved but that the scale used was not sufficiently sensitive to record them at the time. Another study looked at the mood effects of TD compared to a balanced drink in participants with some psychiatric vulnerability (Klaassen et al. 1999). They found no significant effects in participants with no family history of depression but increased depression scores following TD in participants with a family history of depression. There was also a similar trend towards increased anger in the family history positive group.

There have been fewer studies in female subjects. Ellenbogen, Young, Dean, Palmour, and Benkelfat (1996) used a crossover design to examine the effects of two administrations of TD (83 g to adjust for the lower body weight of females and their greater sensitivity to the effects of TD) and a balanced drink given in a counterbalanced order to 20 healthy women. They found increases in negative mood on several scales of the Profile of Mood States (POMS; McNair, Lorr & Droppleman, 1988) and the MRS after the first administration of TD compared to the balanced drink, irrespective of order, but not after the second. Bond, Wingrove, and Critchlow (2001) also used a dose of 83 g and found increased behavioural aggression but no mood changes compared to a control drink. Weltzin, Fernstrom, Fernstrom, Neuberger, and Kaye (1995) used a 100 g drink but also failed to find mood effects in healthy female volunteers, although the group with bulimia nervosa showed increased irritability, which was not related to depression.

Changes in anger after a lower dose drink (50 g) have been more difficult to elicit but a few studies have found them. Knott, Howson, Perugini, Ravindran, and Young (1999) used the POMS to compare changes following 50 mg depletion relative to a balanced control drink in healthy male

volunteers. They found an increase in hostility. In a similar study, Ravindran, Griffiths, Reali, Knott, and Anisman (1999) found a non-significant trend in the same direction. One study (Schmeck et al., 2002) used a smaller mixture of essential amino acids and an unusual control consisting of fruit juice, oatflakes, salt and empty capsules. The participants were divided into four groups of three participants each on the basis of sex and their scores on a German self-rating scale based on the BDHI. Mood was assessed at baseline and following TD using a German adjective checklist. Significant four-way interactions were found for arousal, anger and depression which increased over the day following TD (but not placebo) in the high aggression women only. These findings need to be interpreted cautiously given the small number of participants and somewhat unusual nature of the control condition but are nevertheless suggestive of vulnerability to negative mood effects, including anger, in high aggressive women, confirming the results in men.

6.2.7 Acute Phenylalanine and Tyrosine Depletion

Acute phenylalanine/tyrosine depletion (APTD) is a way of directly manipulating rates of catecholamine synthesis in the brain. A drink containing amino acids including the large neutral amino acids (LNAAs) but excluding tyrosine and phenylalanine thus decreases both dopamine and noradrenaline transmission but there is more evidence for effects on dopamine (Leyton et al., 2004). It thus lacks the specificity to one neurotransmitter that TD has for serotonin. Work with this method of depletion is more recent than that with TD. The drink has been administered in different doses but the dose most often used in healthy controls is 90 g. However, there may be a dose–response relationship which could allow for interesting experimental designs in future studies (Booji et al., 2003).

Some studies have examined the effects of APTD (90 g) on mood in healthy volunteers. Grevet et al. (2002) compared the effects of APTD to a balanced drink in males using a crossover design and found that APTD reduced visual analogue scale ratings of alertness and increased anxiety on the POMS but had no effect on POMS-hostility. Another study with a similar design reported non-specific negative mood effects of a 90 g (20% less for women) drink in a mixed sample of men and women (Harmer, McTavish, Clark, Goodwin, & Cowen, 2001). Subjects felt less good after the depleted drink but no scales related to anger were examined. Lythe, Anderson, Deakin, Elliot, and Strickland (2005) also employed a crossover design and used 10 visual analogue scales including irritability to compare APTD and a balanced drink. They found no mood effects after APTD. McLean, Rubinsztein, Robbins, and Sahakian (2004) employed a parallel groups design in a mixed sample of men and women and found that after APTD, subjects reported less contentment (MRS) and more apathy (on the mean of five visual analogue scales) than after a balanced drink but no measure of anger was employed. These findings indicate some non-specific increases in negative mood but provide no evidence of increases in anger. Two recent studies have examined the effects of APTD in patients recovered from depression but neither found any effects on mood in crossover designs (Roiser et al., 2005; McTavish, Mannie, Harmer, & Cowen, 2005).

6.2.8 Acute Tryptophan Depletion Versus Phenylalanine and Tyrosine Depletion

Two studies have compared the two monamine depletion techniques in healthy volunteers. Leyton et al. (2000) used a parallel group design to compare TD (83 g), APTD and a balanced drink in women. They measured mood on the POMS and VAMS at baseline, 5 h after the drink and after the modified Trier Social Stress Test. TD and APTD both tended to lower mood but this effect was

greater after the stress test. In addition, TD had effects on all six POMS scales including hostility and increased VAMS irritability after the stressful task, whereas APTD only increased bored feelings. (Harrison, Olver, Norman, & Nathan 2002) used a crossover design to compare TD, ATPD and a balanced control and found no evidence of lowered mood but TD did increase fatigue.

Summing up the evidence from the monoamine studies, it appears that both tryptophan and tyrosine depletion can increase vulnerability to increased negative emotions and that this is more likely to occur following exposure to an aversive event. However, only tryptophan depletion has been shown to increase anger and irritability. This would indicate that although several drugs may be effective on improving negative, depressed mood, those with selective action on serotonin should be more effective on symptoms of anger and irritability.

6.2.9 Implications of Neurochemical Work

The evidence from all three types of neurochemical study has supported serotonin as the neurotransmitter most involved in angry aggression and to a lesser extent in the experience of anger itself. Angry reactions are associated with the poor functioning of the 5-HT system. However, biological systems are not discrete entities, and whereas serotonin is an inhibitory neurotransmitter, other neurotransmitters are excitatory influences and these interact. Preclinical work has supported excitatory roles for noradrenaline, dopamine and the endogenous opiates in aggression but little work has been carried out in humans. It may be that the other neurotransmitters are involved in the arousal (noradrenaline) and motor (dopamine) systems necessary to mobilise the response. Drugs acting on different neurotransmitter systems may therefore inhibit or suppress anger through different mechanisms.

6.3 Psychopharmacology of Anger

Anger is commonly reported by psychiatric patients. One large survey found approximately half a sample of 1,300 outpatients reported currently experiencing moderate to severe levels of subjective anger (Posternak & Zimmerman, 2002). This level of anger was found to be comparable to the levels of subjective depression and anxiety reported in the same study. Anger is a symptom of many Axis I disorders such as depression, bipolar disorder, generalised anxiety disorder, post-traumatic stress disorder and premenstrual dysphoric disorder as well as intermittent explosive disorder. It can vary in severity from irritability to extreme rage but it generally improves as the illness remits or is treated. Anger is also a component of many Axis II cluster B disorders such as antisocial, borderline and narcissistic personality disorders. Until recently such disorders were not thought to be amenable to pharmacological treatment but recent studies have proved promising. (Ekselius and Von Knorring 1999) showed that SSRIs can have positive effects on personality traits. Anger can also have a constitutional element as in people who are habitually angry or have a hostile attributional bias but do not meet the criteria for a DSM-IV diagnosis (Wingrove & Bond, 2005). Such anger has a biosocial basis. It may have biological characteristics (Cleare & Bond, 1995, 2000) but may also be routed in adverse social circumstances. It is seldom treated with drugs but some studies have investigated mechanisms in healthy subjects. When anger is a symptom of a clinical disorder, the patient is usually treated for the primary diagnosis but some drugs may be more effective on anger than others or sometimes an additional drug is used to potentiate the effects. The psychopharmacology of anger or irritability as a distressing emotion or symptom will be described first and then the phenomenon of anger attacks, according to the different disorders in which they are likely to appear, will be described.

6.3.1 Anger, Irritability and Hostility

Many people experience increased irritability and anger despite not meeting the criteria for a DSM diagnosis. For example, the incidence of sub-threshold PTSD has been found to be approximately twice that of full PTSD (Kessler, Sonnega, & Bromet, 1995) but to have similar symptoms and high morbidity and mortality. As reported earlier, the experimental work in the neurochemistry of anger has often attempted to recruit people with sub-threshold symptoms by choosing those who score high on a questionnaire measure of anger, irritability or hostility or by employing a task which is likely to increase anger in volunteers. Similarly one drug study has recruited volunteers with sub-threshold symptoms of irritability, who have not sought treatment but have replied to an advertisement and a few others have employed naturalistic designs or novel measures to examine irritability in healthy volunteers. Employing such strategies makes recruitment easier, reduces ethical problems, such as using placebos, decreases drop-out rates and indicates if a drug therapy is likely to be effective in the index clinical group.

6.3.2 Healthy Volunteers

One study recruited volunteers with sub-threshold symptoms of irritability and anxiety and found 5 weeks' treatment with low-dose clomipramine, an antidepressant with serotonergic actions, to lower irritability and anxiety significantly more than treatment with active placebo (Gorenstein, Gentil, Melo, Lotufo-Neto, & Lauriano, 1998). Knutson et al. (1998) administered an SSRI (paroxetine) or placebo to healthy volunteers for 4 weeks. They assessed effects on two subscales (assault and irritability) of the Buss–Durkee Hostility Inventory, the positive and negative affect scales (PANAS) and performance on a collaborative dyadic puzzle task at baseline and after 1 and 4 weeks. They found that ratings of assault and negative affect decreased significantly after both 1 and 4 weeks and irritability after 4 weeks of paroxetine compared to placebo. Affiliative behaviour on the puzzle task increased after 1 week of paroxetine compared to placebo but there was no difference at 4 weeks. In addition, the reduction in negative affect, although not specific to anger, accounted for the reduction in the assault and irritability scales. Tse and Bond (2002) administered 2 weeks' SSRI (citalopram) or placebo to healthy volunteers in a crossover design. They found that citalopram increased assertiveness and they confirmed an increase in cooperative behaviour using a different task but found no effects on mood (PANAS). A fourth study examined the effects of tryptophan (1 g three times a day) compared to placebo administered for 12 days to healthy volunteers in a double-blind, crossover design (Moskowitz, Pinard, Zuroff, Annable, & Young, 2001). They measured self-rated agreeableness-quarrelsomeness and dominance-submission during daily social interactions and found that tryptophan increased self-reported dominance but only decreased quarrelsomeness when it was given after placebo. The authors hypothesised that tryptophan initiated positive change in quarrelsomeness which then persisted to the placebo phase. Tse and Bond (2003) examined the effects of 2 weeks' treatment with a selective noradrenergic reuptake inhibitor (NARI; reboxetine) and placebo in a crossover design. They found no effects on mood (PANAS) but reboxetine increased assertiveness and promoted social bonding. Harmer, Shelley, Cowen and Goodwin (2004) administered citalopram, reboxetine or placebo for 7 days to healthy volunteers in an independent groups design. They examined the effects on mood, hostility and anxiety and on facial expression recognition. They found that both drugs reduced the identification of the anger and fear expressions and increased the relative recall of positive emotional material. In addition, reboxetine decreased hostility.

This work indicates that serotonergic antidepressants not only have positive effects on reducing anger-related emotions but can also increase affiliative or cooperative behaviour and provides some preliminary evidence that a selective noradrenergic antidepressant may also have positive social effects.

6.3.3 Depression

The significance of anger and irritability in different psychiatric disorders is often overlooked (Chapter 27 by R.W. Novaco, this book.) A recent study found that a symptom cluster containing anger, irritability, aggressiveness and hostility was clinically relevant in 23% of a sample of outpatients with a diagnosis of major depressive disorder (Pasquini, Picardi, Biondi, Gaetano, & Morosini, 2004). It is possible from the findings on the neurochemistry of anger that these patients would respond better to serotonergic antidepressants. Serotonin imbalance has previously been linked to a similar cluster but the focus was in relation to impulsive aggressive behaviour rather than angry mood (van Praag, Kahn, & Asnis, 1987; Apter et al., 1990). In order to elucidate the potential advantages of redressing any serotonin dysregulation, patients, who had reported substantial general benefit on SSRI treatment, were asked to retrospectively rate change on a questionnaire (Andrews, Parker and Barrett, 1998). The most marked improvements were shown on irritability, trait depression, worry and neuroticism. This indicates that improvements are not just shown on behaviour but also on angry mood and might be taken to indicate that SSRI treatment would be more effective for depressed patients who report symptoms of anger and irritability. A retrospective analysis examined patients assigned to noradrenergic (desipramine), serotonergic (sertraline, paroxetine) or selective serotonergic and noradrenergic (SNRI; venlafaxine) antidepressants (Bagby et al., 1997). They found that patients classified as high angry hostile did not differ from those classified as low angry hostile in response to the different antidepressant treatments. It is possible that drugs acting on either or both neurotransmitters exert positive effects but through different routes. Drugs acting principally on serotonin might enhance social status and affiliative behaviour, particularly decreasing irritability and aggression, whereas drugs acting principally on noradrenaline might increase drive and energy (Healy and McDonagle, 1997) leading to more social contact and increased social adaptation.

6.3.4 Generalised Anxiety Disorder

Generalised anxiety disorder (GAD) is characterised by psychic symptoms such as anticipatory anxiety, worry, tension and irritability. It is responsive to psychological treatments such as cognitive behavioural therapy. Benzodiazepines used to be the main drug treatment and they are effective but concern over their dependence potential has led to the evaluation of alternative pharmacological treatments. Although tricyclic antidepressants (TCAs) can be effective, their adverse reactions are often not tolerated by these patients who are much more sensitive to bodily side effects than depressed patients. Drugs with serotonergic actions such as buspirone and SSRIs have therefore been evaluated and may be particularly effective for irritability (Wagstaff, Cheer, Matheson, Ormrod, & Goa, 2002). Patients with GAD have a bias to interpret ambiguous information as threatening which can provoke angry as well as anxious responses and one study has shown that, like CBT, SSRIs can significantly reduce threat-related interpretative bias (Mogg, Baldwin, Brodrick, & Bradley, 2004) which would reduce associated emotional responses.

6.3.5 Post-traumatic Stress Disorder

A meta-analysis of 39 studies has shown that anger is a major component of post-traumatic stress disorder (PTSD) (Orth & Wieland, 2006). It forms part of the hyperarousal cluster of symptoms. It has also been suggested that anger may be a mediator for the poor physical health status often found in PTSD (Ouimette, Cronkite, Prins, & Moos, 2004). Psychological therapies, specifically trauma-focused CBT (National Institute for Clinical Excellence, 2005) and eye movement desensitisation and reprocessing (Van Etten & Taylor, 1998) are recognised treatments for PTSD but serotonergic pharmacotherapy is also effective. The SSRIs have been shown to be effective for both short-term and maintenance treatment in a large number of randomised controlled trials (Asnis, Kohn, Henderson, & Brown, 2004). They have not only shown broad clinical effects but have also been shown to reduce symptoms specifically in the hyperarousal cluster. Fluoxetine has been shown to significantly reduce the hyperarousal cluster compared to placebo in RCTs (van der Kolk, Dreyfuss, & Michaels, 1994; Martenyi, Brown, & Zhang, 2002). Sertraline has also been shown to significantly reduce the hyperarousal cluster in two large RCTs (Brady, Pearlstein, & Asnis, 2000; Davidson, Rothbaum, & van der Kolk, 2001). Paroxetine has shown similar effects on the hyperarousal cluster in two large RCTs (Tucker, Zaninelli, & Yehuda, 2001; Marshall, Beebe, & Oldarn, 2001). Other SSRIs and non-selective serotonin potentiating drugs have also shown promising results in open studies. There is little evidence to support the use of other antidepressants in the treatment of anger. Anticonvulsants and buspirone may represent alternative treatments in the future but this evidence is preliminary, although an expert panel has recommended the use of anticonvulsants specifically for symptoms of irritability, anger and impulsivity (Foa, Davidson, & Frances, 1999). Atypical psychotics may be useful in the treatment of anger and paranoia but again the evidence is preliminary (Dillard, Bendfeldt, & Jernigan, 1993). Benzodiazepine treatment (alprazolam) has not been found to be effective in one RCT (Braun, Greenberg, & Dasberg, 1990). In addition, when administered soon after acute trauma to evaluate their efficacy as preventative treatment, benzodiazepines were not found to prevent PTSD from developing (Asnis et al., 2004). However, some recent work decreasing noradrenergic output has produced some promising preliminary results. It has been shown that an increase of noradrenergic activity during a life-threatening event contributes to the strengthening of memory for the trauma, resulting in distressing symptoms. Preventing pre-synaptic noradrenaline release with alpha-2-adrenergic agonists, or blocking post-synaptic noradrenaline receptors with beta-adrenergic antagonists such as propranolol, should reduce these enhanced memories and fear conditioning. Propranolol administered shortly after the event has been shown to be an effective treatment (Pitman & Delahanty, 2005) and is therefore likely to decrease anger as a secondary effect.

6.3.6 Premenstrual Dysphoric Disorder

Irritability is a core symptom of premenstrual dysphoric disorder (PMDD) in DSM-IV (APA 1994) and irritability, anger and internal tension are the symptoms most likely to lead to treatment seeking. The aetiology of PMDD is considered to involve several systems including the hypothalamic-pituitary-gonadal (HPG) axis and neurotransmitters. Normal ovarian function triggers biochemical events in the brain and peripherally which lead to the changes, including fluctuations in mood, associated with the menstrual cycle. These changes are recognised by most women but for a small number they represent a severe disorder. The HPG axis is closely linked to the neurotransmitter serotonin (5-HT) (Steiner & Pearlstein, 2000) and increasing evidence implicates 5-HT dysregulation in PMDD (Kouri & Halbreich, 1997). SSRIs have not only been found to be particularly

effective for this disorder (Dimmock, Wyatt, Jones, & O'Brien, 2000; Steiner & Born, 2000) but also to be more effective than tricyclic antidepressants (Freeman, Rickels, Sondheim, & Polansky, 1999). This efficacy appears to differ from their antidepressant action. The response to treatment is more rapid than when SSRIs are used for other disorders such as depression, panic disorder or obsessive compulsive disorder, and intermittent treatment has been shown to be as effective as continuous dosing (Wikander, Sunblad, & Andersch, 1998).

6.3.7 Bipolar Disorder

Irritability is a core symptom of bipolar-II disorder and may be a useful marker of the disorder (Benazzi, 2006). However, monotherapy of anger or irritability in bipolar disorder with SSRIs or other antidepressants is not recommended as this can promote a switch into a hypomanic state (Kupfer, Carpenter, & Frank, 1988; Kusumaker, 2002). Mood stabilisers such as lithium or anticonvulsants such as carbamazepine, gabapentin, sodium valproate or divalproex sodium are effective (Moller, Grunze, & Broich, 2006). In a study specifically examining effects on mood, divalproex sodium was shown to reduce anger–hostility on the SCL-90 significantly more than placebo in female patients comorbid for bipolar disorder and borderline personality disorder (Frankenburg & Zanarini, 2002).

6.3.8 Axis II Disorders

Axis II cluster B personality disorders are characterised by both anger and aggression. Patients diagnosed with borderline personality disorder (BPD), in particular, exhibit intense anger and emotional instability. It has been suggested that both SSRIs and atypical antipsychotics alleviate anger and irritability in these patients (Zanarini, 2004) but most of the evidence comes from uncontrolled trials. One trial examined the response of patients with major depressive disorder who had an additional cluster B personality disorder and found they responded better to fluoxetine than nortriptyline (Mulder, Joyce, & Luty, 2003). Another study in female patients with BPD, which controlled for Axis I disorders statistically, found that although fluvoxamine improved mood stability, it was no more effective than placebo on anger (Rinne, van den Brink, Wouters, & van Dyke, 2002). A few RCTs have been conducted with SSRIs in patients without comorbid Axis I disorders. In these trials, fluoxetine has been found to improve anger in symptomatic volunteers with BPD (Salzman et al., 1995) and to improve irritability in patients with mixed personality disorders (Coccaro & Kavoussi, 1997).

Anticonvulsants have been shown to be effective in many uncontrolled and a few controlled trials. Divalproex sodium was shown to reduce anger–hostility on the SCL-90 significantly more than placebo in female patients comorbid for BPD and bipolar disorder (Frankenburg & Zanarini, 2002). Topiramate reduced anger on 4 of the 5 scales of the Spielberger trait anger expression scale (STAXI) compared to placebo in male patients with BPD (Nickel, Nickel, & Kaplan, 2005). This efficacy was confirmed in a recent RCT comparing the anticonvulsant, lamotrigine, to placebo in women with BPD (Tritt et al., 2005). Lamotrigine was shown to reduce anger on 4 scales of the 5 STAXI scales. The only scale which did not show a significant effect was Anger-In which measures a tendency to repress anger. However, de la Fuente and Lotstra (1994) found no significant improvement on the Hopkins Symptom Checklist-90 (SCL-90) after carbamazepine compared to placebo in BPD patients.

Antipsychotic agents have also been shown to reduce irritability in patients with BPD. Haloperidol was compared to amitriptyline and placebo (Soloff, George, & Nathan, 1989) and to phenelzine and placebo (Soloff, Cornelius, & George, 1993) and in both RCTs was found to be significantly more effective than placebo in reducing hostility measured by the SCL-90. The atypical antipsychotic, olanzapine has also been found to reduce anger–hostility on the SCL-90 compared to placebo in female patients with BPD (Zanarini & Frankenberg, 2001). It is unfortunate that hardly any trials have compared SSRIs to anticonvulsants or antipsychotics and it should be remembered that the adverse event profiles of SSRIs are considerably more benign than these alternatives.

6.3.9 Anger Outbursts or Attacks

Although irritability can precede anger attacks, Winkler et al. (2006b) found that it was only weakly associated with them. Much clearer relationships were shown with overreaction to minor annoyances and episodes of inappropriate anger or rage. Thus the authors suggested that one or two simple screening questions based on these items could improve diagnosis. Although anger outbursts can occur in different disorders, they have been described most clearly with respect to depression.

6.3.9.1 Anger Attacks in Depression

Anger attacks occurring as part of major depressive disorder (MDD) have been described comprehensively (Fava et al., 1993; Rosenbaum et al., 1993). These attacks are very similar to panic attacks. They are accompanied by symptoms of autonomic activation such as tachycardia, sweating, flushing, and tightness of the chest but the overriding emotion is anger and not the fear or anxiety that accompanies panic attacks. The attacks are experienced as ego-dystonic and inappropriate to the situations in which they occur. It has been estimated that approximately one third of patients with unipolar depression present with anger attacks, and therefore it has been proposed that unipolar depression with anger attacks represents a unique subtype of depression (Fava & Rosenbaum, 1998). However, it should be noted that these patients are also more likely to meet criteria for Axis II personality disorders than depressed patients without anger attacks (Tedlow et al., 1999). It has been suggested that depressed patients who report anger attacks may have greater central serotonergic dysregulation than those not reporting attacks and would therefore respond preferentially to SSRIs (Fava et al., 1993). The hypothesis was confirmed by blunted prolactin responses to a fenfluramine challenge in depressed patients with anger attacks (Fava et al., 2000) and by decreased 5HT₂ receptor binding potential after 6 weeks' treatment with the serotonergic antidepressant, nefazodone (Mischoulon et al., 2002). However, patients with anger attacks show similar response rates to antidepressant treatments as patients without them and anger attacks subside after treatment with both SSRIs and tricyclic antidepressants in 53–71% (Fava et al., 1997). Therefore antidepressants with both serotonergic and noradrenergic actions have been shown to decrease this form of anger.

6.3.9.2 Anger Attacks in Other Disorders

Anger attacks have been less studied in other Axis I disorders and there are few reports of controlled treatment studies. However, they have recently been identified in patients with seasonal affective disorder (SAD). SAD is a variant of recurrent depression. The prevalence is similar to rates in depression but patients with SAD report more frequent attacks and more autonomic symptoms and behavioural outbursts and so they appear to experience the attacks as more intense (Winkler, Pjrek,

Kindler, Heiden, & Kasper, 2006a). In general, these patients respond well to treatment with bright artificial light or antidepressants (Pjrek, Winkler, & Kasper, 2005). Although there are no studies focusing on the treatment of anger attacks, they are likely to respond to selective antidepressants.

Although anger attacks also occur in *anxiety* disorders including panic disorder, the prevalence has been shown to be lower than in those with a current or past history of depression (Gould et al., 1996). SSRIs are likely to be effective as they have shown efficacy in many types of anxiety (Wagstaff et al., 2002).

Anger attacks are also common in *PTSD*. One study assessed the impact of exposure to a trauma in male college students (Jakupcak & Tull, 2005). Those who reported symptoms of PTSD had higher trait anger and hostility and more aggression than those not reporting symptoms. As SSRIs are the preferred treatment for PTSD (Asnis et al., 2004) and anger attacks are likely to be an integral part of the disorder, they are likely to respond to conventional treatment. However, immediate treatment with propranolol may prevent their occurrence.

Anger attacks also occur in *bipolar depression*. Perlis et al. (2004) examined the prevalence in patients with unipolar depression compared to the depressed phase in bipolar depression. They found anger attacks to be more common in the bipolar patients (62%) than the depressed patients (26%) and the presence of anger attacks predicted bipolarity. However, the reported incidence in another study was only 12% and so Mammen, Pilkonis, Chengappa and Kupfer (2004) investigated the presence of anger attacks in 45 bipolar patients and their response to treatment with citalopram added to a mood stabiliser. They found 38.6% of the patients reported anger attacks and that these reduced significantly after citalopram. Interestingly, trait anger, not hypomanic or depressive symptoms, predicted the presence of anger attacks, suggesting that anger attacks may not be a manifestation of hypomanic symptoms. The rate of anger attacks was similar to that found in major depression. Although uncontrolled, this study indicates that conventional treatment for bipolar disorder would be insufficient to combat anger attacks and SSRI treatment would be more effective.

Anger attacks can occur in the absence of any other psychiatric disorder and therefore can meet the diagnostic criteria for *intermittent explosive disorder* (IED) of which they are a cardinal symptom. However, some work has linked IED to bipolar disorder (McElroy, 1999). This work found that anger attacks in individuals, who met the DSM-IV criteria for IED, were associated with manic-like affective symptoms and a high rate of comorbid lifetime bipolar disorder. Lithium is the recommended treatment for mania (Licht, 1998). Interestingly, lithium has been shown to be effective in the treatment of emotionally charged aggression (Sheard, 1975) and IED has been shown to respond to mood-stabilising drugs (McElroy, 1999).

Anger outbursts are associated with cluster B *personality disorders* and form part of the diagnosis. However, there may be a qualitative difference with anger attacks described in depression. Although the outbursts are accompanied by autonomic activation and are viewed as inappropriate reactions by others, there is little evidence that they are seen as ego-dystonic by the patients who may think their reactions are justified. They might also be described as aggression rather than anger. With these provisos, some pharmacological treatments will be described. Although serotonergic antidepressants, such as fluoxetine, have been found to improve aggressive reactions in patients with mixed personality disorders (Coccaro & Kavoussi, 1997), other drugs have also been found to be effective on these outbursts. Anticonvulsants such as divalproex sodium have been shown to reduce behavioural expressions of anger on the overt aggression scale (OAS) in BPD patients (Hollander, Allen and Lopez, 2001) in cluster B personality disorders (Hollander, Tracy and Swann, 2003) and in female patients comorbid for BPD and bipolar disorder (Frankenburg and Zanarini, 2002). Similarly, in a recent RCT comparing the anticonvulsant, lamotrigine, to placebo in women with BPD (Tritt et al., 2005), lamotrigine was shown to reduce outbursts and to help control. In addition, olanzapine has also been evaluated in combination with fluoxetine and was found to be superior

to fluoxetine alone in reducing behavioural aggression measured on the OAS in women with BPD (Zanarini, Frankenburg, & Parachini, 2004). Therefore aggressive outbursts in these patients may respond better to anticonvulsant or antipsychotic treatments.

6.4 Conclusions

This review reveals that most work in the neurochemistry of anger has focussed on serotonin. Noradrenaline appears to be important in reactions to distressing experiences as in PTSD but this may be more important in the generation of anxiety rather than anger.

Both the experimental neurochemical work and the psychopharmacology point to the importance of individual differences in trait aggression, irrespective of diagnosis, in the experience and display of anger. The work on both neurotransmitter metabolites in CSF and neuroendocrine challenges has specifically examined trait irritability and hostility in different diagnostic groups and found them to be associated with hypofunctioning of the serotonergic system and, to a lesser extent, hyperfunctioning of the noradrenergic system. The work with monoamine depletion also demonstrates a vulnerability to the negative effects of tryptophan depletion on anger in both men and women with a hostile disposition. In contrast, participants with low levels of trait hostility were found not to respond negatively to tryptophan depletion. Either their biological system was able to correct the deficit soon after the challenge or their cognitive interpretation of any changes experienced did not involve negative feelings like anger. In addition, the finding that individual differences in trait aggression predicted the presence of anger attacks in bipolar disorder (Mammen et al., 2004) indicates that personality is at least as important a variable as illness in the presentation of anger. Therefore individual differences interact with biological characteristics in the likelihood of anger occurring or being displayed.

As well as individual differences, another important variable is exposure to threat or adversity. Anger has many causes but the most common is a sense of injustice or unfairness resulting from a real or perceived transgression or infringement (Velasco & Bond, 1998; Chapter 15 by T. Wranik & K.R. Scherer, this book). Anger occurring as part of PTSD is likely to result from the attributed cause of a trauma. Noradrenaline has been associated with conditioning to the immediate fear reaction to the threat and the physiological responses but anger may occur later in the processing cycle. Monoamine depletion was found to be more likely to increase negative emotions following exposure to an aversive event. Only tryptophan depletion has been found to increase anger but these studies have used more provocative experimental tasks. Patients with cluster B personality disorders display interpersonal difficulties because they overreact to minor events. In neuroendocrine challenge studies, irritability in these patients was inversely related to serotonergic and positively related to noradrenergic functioning. Anger attacks have been shown to be ego-dystonic and to represent an overreaction to minor annoyances. Drugs acting on both serotonin and noradrenaline have been shown to reduce responses to threat. Citalopram was shown to reduce threat-related interpretative bias in GAD and propranolol was shown to reduce symptoms of PTSD. In fact propranolol has both 5-HT¹ and 5-HT² antagonist properties. A reduction in cognitive and physiological responses to threat is likely to reduce irritability and anger.

As well as the association between deficiencies in serotonin and anger, positive associations have been found between PRL responses to fenfluramine and both conscientiousness and positive mood. These findings indicate that a well-functioning 5-HT system is involved in mood regulation. Work in healthy volunteers has not only shown tryptophan enhancement and SSRIs to be associated with decreases in anger but also to increase cooperative and affiliative behaviour. Reboxetine has also been shown to have prosocial effects. Thus, these selectively acting drugs do not just work as

anti-depressants but have more wide-ranging positive social effects. However, as neurotransmitter systems are inextricably linked, the balance between different neurochemical systems is likely to be important in healthy functioning.

The psychopharmacology of anger supports the neurochemistry findings but suffers from a lack of well-controlled clinical trials. Because anger is a symptom and not a disorder, except in the case of IED, it is rarely the focus of treatment. However, it is apparent that SSRIs have a wide profile of effects and evidence exists for their efficacy in many Axis I disorders in which irritability and anger are core symptoms. The exception is bipolar disorder in which mood stabilisers are indicated. However, both lithium and carbamazepine enhance 5-HT activity. There is also some evidence for efficacy in Axis II personality disorders but there are very few comparative trials with anticonvulsants or antipsychotics which have also shown efficacy. However, antipsychotics are not selective drugs and many block 5-HT² receptors in addition to their anti-dopaminergic effects. The relatively benign adverse event profile of SSRIs should make them the first-line treatment, reserving other drugs for non-responsive cases.

References

- American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: American Psychiatric Association.
- Andrews, W., Parker, G., & Barrett, E. (1998). The SSRI antidepressants: Exploring their "other" possible properties. *Journal of Affective Disorders*, 49, 141–144.
- Apter, A., van Praag, H. M., Plutchik, R., Sevy, S., Korn, M., & Brown, S.-L. (1990). Interrelationships among anxiety, aggression, impulsivity, and mood: A serotonergically linked cluster? *Psychiatry Research*, 32, 191–199.
- Archer, J. (2006). The relation between scores on the buss-perry aggression questionnaire and aggressive acts, impulsiveness, competitiveness, dominance, and sexual jealousy. *Aggressive Behavior*, 32, 464–473.
- Asnis, G. M., Kohn, S. R., Henderson, M., & Brown, N. L. (2004). SSRIs versus non-SSRIs in post-traumatic stress disorder: An update with recommendations. *Drugs*, 64, 383–404.
- Bagby, R. M., Kennedy, S. H., Schuller, D. R., Dickens, S. E., Minifie, C. E., Levitt, A., et al. (1997). Differential pharmacological treatment response in high angry hostile and low angry hostile depressed patients: A retrospective analysis. *Journal of Affective Disorders*, 45, 161–166.
- Bandura, A. (1973). *Aggression: A social learning analysis*. Upper Saddle River, NJ: Prentice-Hall.
- Baron, R. A. (1977). *Human aggression*. New York and London: Plenum Press.
- Baumeister, R. F. (1994). *Losing control*. San Diego, CA: Academic Press.
- Benazzi, F. (2006). Symptoms of depression as possible markers of bipolar II disorder. *Progress in Neuro-Psychopharmacology & Biological Psychiatry*, 30, 471–477.
- Bond, A. J., & Lader, M. H. (1974). The use of analogue scales in rating subjective feelings. *British Journal of Medical Psychology*, 47, 211–218.
- Bond, A. J., & Lader, M. H. (1986). A method to elicit aggressive feelings and behaviour via provocation. *Biological Psychology*, 22, 69–79.
- Bond, A. J., Ruaro, L., & Wingrove, J. (2006). Reducing anger induced by ego threat: Use of vulnerability expression and influence of trait characteristics. *Personality and Individual Differences*, 40, 1087–1097.
- Bond, A. J., Wingrove, J., & Critchlow, D. G. (2001). Tryptophan depletion increases aggression in women in the premenstrual phase. *Psychopharmacology*, 156, 477–480.
- Booji, L., Van der Does, A. J. W., & Riedel, W. J. (2003). Monoamine depletion in psychiatric and healthy populations: Review. *Molecular Psychiatry*, 8, 951–973.
- Brady, K., Pearlstein, T., Asnis, G. M., et al. (2000). Efficacy and safety of sertraline treatment of posttraumatic stress disorder: A randomized controlled trial. *JAMA*, 283, 1837–1844.
- Braun, B. P., Greenberg, D., Dasberg, D. H., et al. (1990). Core symptoms of posttraumatic stress disorder unimproved by alprazolam treatment. *Journal of Clinical Psychiatry*, 51, 236–238.
- Brown, G. L., & Goodwin, F. K. (1984). Diagnostic, clinical and personality characteristics of aggressive men with low CSF 5-HIAA. *Clinical Neuropharmacology*, 7, S.
- Bushman, B. J., & Anderson, C. A. (2001). Is it time to pull the plug on the hostile versus instrumental aggression dichotomy? *Psychological Review*, 108, 273–279.

- Buss, A. H., & Durkee, A. (1957). An inventory for assessing different kinds of hostility. *Journal of Consulting Psychology*, 21, 343–349.
- Buss, A. H., & Perry, M. (1992). The aggression questionnaire. *Journal of Personality and Social Psychology*, 59, 73–81.
- Cleare, A. J., & Bond, A. J. (1994). Effects of alterations in plasma tryptophan levels on aggressive feelings. *Archives of General Psychiatry*, 51, 1004–1005.
- Cleare, A. J., & Bond, A. J. (1995). The effect of tryptophan depletion and enhancement on subjective and behavioural aggression in normal male subjects. *Psychopharmacology*, 118, 72–81.
- Cleare, A. J., & Bond, A. J. (1997). Does central serotonergic function correlate inversely with aggression? A study using d-fenfluramine in healthy subjects. *Psychiatry Research*, 69, 89–95.
- Cleare, A. J., & Bond, A. J. (2000). Ipsapirone challenge in aggressive men shows an inverse correlation between 5-HT_{1A} receptor function and aggression. *Psychopharmacology*, 148, 344–349.
- Coccaro, E. F., Gabriel, S., & Siever, L. J. (1990). Buspirone challenge: Preliminary evidence for a role for central 5-HT_{1A} receptor function in impulsive aggressive behavior in humans. *Psychopharmacology Bulletin*, 26, 393–405.
- Coccaro, E. F., & Kavoussi, R. J. (1996). Neurotransmitter correlates of impulsive aggression. In D. M. Stoff & R. B. Cairns (Eds.), *Aggression and violence* (pp. 67–85). Mahwah, NJ: Lawrence Erlbaum Associates.
- Coccaro, E. F., & Kavoussi, R. J. (1997). Fluoxetine and impulsive aggressive behavior in personality-disordered subjects. *Archives of General Psychiatry*, 54, 1081–1088.
- Coccaro, E. F., Lawrence, T., Trestman, R., Gabriel, S., Klar, H. M., & Siever, L. J. (1991). Growth hormone responses to intravenous clonidine challenge correlate with behavioral irritability in psychiatric patients and healthy volunteers. *Psychiatry Research*, 39, 129–139.
- Coccaro, E. F., Siever, L. J., Owen, K., & Davis, K. L. (1989). Serotonergic studies in patients with affective and personality disorders. *Archives of General Psychiatry*, 47, 587–599.
- Cook, W. W., & Medley, D. M. (1954). Proposed hostility and pharisaic-virtue scales for the MMPI. *Journal of Applied Psychology*, 38, 414–418.
- Coote, M., Wilkins, A., Werstiuik, E. S., & Steiner, M. (1998). Effects of electroconvulsive therapy and desipramine on neuroendocrine responses to the clonidine challenge test. *Journal of Psychiatry and Neuroscience*, 23, 172–178.
- Danjou, P., Hamon, M., Lacomblez, L., Warot, D., Kecskemeti, S., & Puech, A. J. (1990). Psychomotor, subjective and neuroendocrine effects of acute tryptophan depletion in the healthy volunteer. *Psychiatry and Psychobiology*, 5, 31–38.
- Davidson, R. J., Jackson, D. C., & Kalin, N. H. (2000). Emotion, plasticity, context, and regulation: Perspectives from affective neuroscience. *Psychological Bulletin*, 126, 890–909.
- Davidson, J. R., Rothbaum, B. O., van der Kolk, B. A., et al. (2001). Multicenter, double-blind comparison of sertraline and placebo in the treatment of posttraumatic stress disorder. *Archives of General Psychiatry*, 58, 485–492.
- de la Fuente, I. M., & Lotstra, F. (1994). A trial of carbamazepine in borderline personality disorder. *European Neuropsychopharmacology*, 4, 479–486.
- Dillard, M. I., Bendfeldt, F., & Jernigan, P. (1993). Use of thioridazine in posttraumatic stress disorder. *Southern Medical Journal*, 86, 1276–1278.
- Dimmock, P. W., Wyatt, K. M., Jones, P. W., & O'Brien, P. M. S. (2000). Efficacy of selective serotonin-reuptake inhibitors in premenstrual syndrome: A systematic review. *Lancet*, 356, 1131–1136.
- Dodge, K. A., & Coie, J. D. (1987). Social-information-processing factors in reactive and proactive aggression in children's peer groups. *Journal of Personality and Social Psychology*, 53, 1146–1158.
- Ekselius, L., & Von Knorring, L. (1999). Changes in personality traits during treatment with sertraline or citalopram. *British Journal of Psychiatry*, 174, 444–448.
- Ellenbogen, M. A., Young, S. N., Dean, P., Palmour, R. M., & Benkelfat, C. (1996). Mood response to acute tryptophan depletion in healthy volunteers; sex differences and temporal stability. *Neuropsychopharmacology*, 15, 465–474.
- Fava, M., Nierenberg, A. A., Quitkin, F. M., Zisook, S., Pearlstein, T., Stone, A., et al. (1997). A preliminary study of the efficacy of sertraline and imipramine on anger attacks in atypical depression and dysthymia. *Psychopharmacology Bulletin*, 33, 101–103.
- Fava, M., Rosenbaum, A. F., Fava, J. A., McCarthy, M. K., Steingard, R. J., & Bouffides, E. (1993). Anger attacks in unipolar depression, Part 1: Clinical correlates and response to fluoxetine treatment. *American Journal of Psychiatry*, 150, 1158–1163.
- Fava, M., & Rosenbaum, J. F. (1998). Anger attacks in depression. *Depression & Anxiety* 8, Suppl. 1, 59–63.
- Fava, M., Vuolo, R. D., Wright, E. C., Nierenberg, A. A., Alpert, J. E., & Rosenbaum, J. F. (2000). Fenfluramine challenge in unipolar depression with and without anger attacks. *Psychiatry Research*, 94, 9–18.
- Finn, P. R., Young, S. N., Pihl, R. O., & Ervin, F. R. (1998). The effects of acute plasma tryptophan manipulation on hostile mood: The influence of trait hostility. *Aggressive Behavior*, 24, 173–185.

- Foa, E. B., Davidson, J. R. T., & Frances, A. (1999). The expert consensus guideline series: treatment of posttraumatic stress disorder: The Expert Consensus Panels for PTSD. *Journal of Clinical Psychiatry*, 60, Suppl. 16, 1–75.
- Flory, J. D., Manuck, S. B., Matthews, K. A., & Muldoon, M. F. (2004). Serotonergic function in the central nervous system is associated with daily ratings of positive mood patients and healthy volunteers. *Psychiatry Research*, 129, 11–19.
- Frankenburg, F., & Zanarini, M. (2002). Divalproex sodium treatment of women with borderline personality disorder and bipolar II disorder: A double blind, placebo controlled pilot study. *Journal of Clinical Psychiatry*, 63, 442–446.
- Freeman, E. W., Rickels, K., Sondheim, S. J., & Polansky, M. (1999). Differential response to antidepressants in women with premenstrual syndrome/premenstrual dysphoric disorder: A randomised controlled trial. *Archives of General Psychiatry*, 56, 932–939.
- Gijssman, H. J., Van Gerven, J. M. A., Tieleman, M. C., Schoemaker, R. C., Pieters, M. S. M., Ferrari, M. D., Cohen, A. F., & Van Kempen, G. M. J. (1998). Pharmacokinetic and pharmacodynamic profile of oral and intravenous metachlorophenylpiperazine in healthy volunteers. *Journal of Clinical Psychopharmacology*, 18, 289–295.
- Gorenstein, C., Gentil, V., Melo, M., Lotufo-Neto, F., & Lauriano, V. (1998). Mood improvement in “normal” volunteers. *Journal of Psychopharmacology*, 12, 246–251.
- Gould, R. A., Ball, S., Kaspi, S. P., Otto, M. W., Pollack, M. H., Shekhar, A., et al. (1996). Prevalence and correlates of anger attacks: A two site study. *Journal of Affective Disorders*, 39, 31–38.
- Grevet, E. H., Tietzmann, M. R., Shansis, F. M., Hastenpflugl, C., Santana, L. C., Forster, L., et al. (2002). Behavioural effects of acute phenylalanine and tyrosine depletion in healthy male volunteers. *Journal of Psychopharmacology*, 16, 51–55.
- Harmer, C. J., McTavish, S. F., Clark, L., Goodwin, G. M., & Cowen, P. J. (2001). Tyrosine depletion attenuates dopamine function in healthy volunteers. *Psychopharmacology*, 154, 105–111.
- Harmer, C. J., Shelley, N. C., Cowen, P. J., & Goodwin, G. M. (2004). Increased positive versus negative affective perception and memory in healthy volunteers following selective serotonin and norepinephrine reuptake inhibition. *American Journal of Psychiatry*, 161, 1256–1263.
- Harrison, B. J., Olver, J. S., Norman, T. R., & Nathan, P. J. (2002). Effects of serotonin and catecholamine depletion on interleukin-6 activation and mood in human volunteers. *Human Psychopharmacology*, 17, 293–7.
- Healy, D., & McMonagle, T. (1997). The enhancement of social functioning as a therapeutic principle in the management of depression. *Journal of Psychopharmacology*, 11(Suppl), S25–S31.
- Hollander, E., Allen, A., Lopez, R. P., et al. (2001). A preliminary, double-blind, placebo-controlled trial of divalproex in borderline personality disorder. *Journal of Clinical Psychiatry*, 62, 199–203.
- Hollander, E., Tracy, K. A., Swann, A. C., et al. (2003). Divalproex in the treatment of impulsive aggression: efficacy in cluster B personality disorders. *Neuropsychopharmacology*, 28, 1186–1197.
- Jakupcak, M., & Tull, M. T. (2005). Effects of trauma exposure on anger, aggression, and violence in a nonclinical sample of men. *Violence and Victims*, 20, 589–598.
- Kahn, R. S., & Wetzler, S. (1991). m-chlorophenylpiperazine as a probe of serotonin function. *Biological Psychiatry*, 30, 1139–1166.
- Kessler, R. C., Sonnega, A., Bromet, E., et al. (1995). Posttraumatic stress disorder in the National Comorbidity Study. *Archives of General Psychiatry*, 52, 1048–1060.
- Klaassen, T., Riedel, W. J., van Praag, H. M., Menheere, P. P. C. A., & Griez, E. (2004). Neuroendocrine response to meta-chlorophenylpiperazine and ipsapirone in relation to anxiety and aggression. *Psychiatry Research*, 113, 29–40.
- Klaassen, T., Riedel, W. J., van Someren, A., Deutz, N. E. P., Honig, A., & van Praag, H. M. (1999). Mood effects of 24-hour tryptophan depletion in healthy first-degree relatives of patients with affective disorders. *Biological Psychiatry*, 46, 489–497.
- Knott, V. J., Howson, A. L., Perugini, M., Ravindran, A. V., & Young, S. N. (1999). The effect of acute tryptophan depletion and fenfluramine on quantitative EEG and mood in healthy male subjects. *Biological Psychiatry*, 46, 229–238.
- Knutson, B., Wolkowitz, O. M., Cole, S. W., Chan, T., Moore, E. A., Johnson, R. C., et al. (1998). Selective alteration of personality and social behavior by serotonergic intervention. *American Journal of Psychiatry*, 155, 373–379.
- Kouri, E. M., & Halbreich, U. (1997). State and trait serotonergic abnormalities in women with dysphoric premenstrual syndromes. *Psychopharmacology Bulletin*, 33, 767–770.
- Kupfer, D. I., Carpenter, L. L., & Frank, E. (1988). Possible role of antidepressants in precipitating mania and hypomania in recurrent depression. *American Journal of Psychiatry*, 145, 804–808.
- Kusumaker, V. (2002). Antidepressants and antipsychotics in the long-term treatment of bipolar disorder. *Journal of Clinical Psychiatry*, 63(Suppl. 10), 23–28.
- Lester, D. (1995). The concentration of neurotransmitter metabolites in the cerebrospinal fluid of suicidal individuals: A meta-analysis. *Pharmacopsychiatry*, 28, 45–50.

- Leyton, M., Dagher, A., Boileau, I., Casey, K., Baker, G. B., Ddiksic, M., et al. (2004). Decreasing amphetamine-induced dopamine release by acute phenylalanine/tyrosine depletion: A PET/[11C]raclopride study in healthy men. *Neuropsychopharmacology*, 29, 427–432.
- Leyton, M., Young, S. N., Pihl, R. O., Etezadi, S., Lauze, C., Blier, P., et al. (2000). Effects on mood of acute phenylalanine/tyrosine depletion in healthy women. *Neuropsychopharmacology*, 22, 52–63.
- Licht, R. W. (1998). Drug treatment of mania: A critical review. *Acta Psychiatrica Scandinavica*, 97, 387–397.
- Linnoila, M., Virkkunen, M., Scheinin, M., Nuutila, A., Rimon, R., & Goodwin, F. K. (1983). Low cerebrospinal fluid 5-hydroxyindoleacetic acid concentration differentiates impulsive from nonimpulsive violent behaviour. *Life Sciences*, 33, 2609–2614.
- Lythe, K. E., Anderson, I. M., Deakin, J. F. W., Elliot, R., & Strickland, P. L. (2005). Lack of behavioural effects after acute tyrosine depletion in healthy volunteers. *Journal of Psychopharmacology*, 19, 5–11.
- Mammen, O. K., Pilkonis, P. A., Chengappa, K. N., & Kupfer, D. J. (2004). Anger attacks in bipolar depression: Predictors and response to citalopram added to mood stabilizers. *The Journal of clinical psychiatry*, 65, 627–633.
- Mann, J. J., Waternaux, C., Haas, G. L., & Malone, K. M. (1999). Towards a clinical model of suicidal behaviour in psychiatric patients. *American Journal of Psychiatry*, 156, 181–189.
- Manuck, S. B., Flory, J. D., McCaffery, J. K., Matthews, K. A., Mann, J. J., & Muldoon, M. F. (1998). Aggression, impulsivity, and central nervous system serotonergic responsivity in a nonpatient sample. *Neuropsychopharmacology*, 19, 287–299.
- Manuck, S. B., Flory, J. D., Muldoon, M. F., & Ferrell, R. D. (2002). Central nervous system serotonergic responsivity and aggressive disposition in men. *Physiology and Behaviour*, 77, 705–709.
- Marshall, R. D., Beebe, K. L., Oldam, M., et al. (2001). Efficacy and safety of paroxetine treatment for chronic PTSD: A fixed-dose, placebo-controlled study. *American Journal of Psychiatry*, 158, 1982–1988.
- Marshall, R. D., Blanco, C., Printz, D., Liebowitz, M. R., Klein, D. F., & Coplan, J. (2002). A pilot study of noradrenergic and HPA axis functioning in PTSD vs. panic disorder. *Psychiatry Research*, 110, 219–230.
- Martenyi, F., Brown, E. B., Zhang, H., et al. (2002). Fluoxetine versus placebo in posttraumatic stress disorder. *Journal of Clinical Psychiatry*, 63, 199–206.
- McElroy, S. L. (1999). Recognition and treatment of DSM-IV intermittent explosive disorder. *Journal of Clinical Psychiatry*, 60(Suppl. 15), 12–16.
- McLean, A., Rubinsztein, J. S., Robbins, T. W., & Sahakian, B. J. (2004). The effects of tyrosine depletion in normal healthy volunteers: Implications for unipolar depression. *Psychopharmacology*, 171, 286–297.
- McNair, D. M., Lorr, M., & Droppleman, L. F. (1988). San Diego, Educational and Industrial Testing Service. *Manual for the Profile of Mood States*.
- McTavish, S. F., Mannie, Z. N., Harmer, C. J., & Cowen, P. J. (2005). Lack of effect of tyrosine depletion on mood in recovered depressed women. *Neuropsychopharmacology*, 30, 786–791.
- Mischoulon, D., Dougherty, D. D., Bottonari, K. A., Gresham, R. L., Sonawalla, S. B. B., Fischman, A. J., et al. (2002). An open pilot study of nefazodone in depression with anger attacks: Relationship between clinical response and receptor binding. *Psychiatry Research*, 116, 151–161.
- Mogg, K., Baldwin, D. S., Brodrick, P., & Bradley, B. P. (2004). Effect of short-term SSRI treatment on cognitive bias in generalised anxiety disorder. *Psychopharmacology*, 176, 466–470.
- Moller, H. J., Grunze, H., & Broich, K. (2006). Do recent efficacy data on the drug treatment of acute bipolar depression support the position that drugs other than antidepressants are the treatment of choice? A conceptual review. *European Archives of Psychiatry and Clinical Neuroscience*, 256, 1–16.
- Moller, S. E., Mortensen, E. L., Breum, L., Alling, C., Larsen, O. G., Boge-Rasmussen, T., et al. (1996). Aggression and personality: Association with amino acids and monoamine metabolites. *Psychological Medicine*, 26, 323–331.
- Moskowitz, D. S., Pinard, G., Zuroff, D. C., Annable, L., & Young, S. N. (2001). The effect of tryptophan on social interaction in everyday life: A placebo-controlled study. *Neuropsychopharmacology*, 25, 277–289.
- Mulder, R. T., Joyce, P. R., & Luty, S. E. (2003). The relationship of personality disorders to treatment outcome in depressed outpatients. *Journal of Clinical Psychiatry*, 64, 259–64.
- National Institute for Clinical Excellence (2005) Post-traumatic stress disorder. The management of PTSD in adults and children in primary and secondary care. National Clinical Practice Guideline number 26. The Royal College of Psychiatrists and the British Psychological Society, London.
- New, A. S., Trestman, R. F., Mitropoulou, V., Goodman, M., Koenigsberg, H. H., Silverman, J., et al. (2004). Low prolactin response to fenfluramine in impulsive aggression. *Journal of Psychiatric Research*, 38, 223–230.
- Newman, M. E., Shapira, B., & Lerer, B. (1998). Evaluation of central serotonergic function in affective and related disorders by the fenfluramine challenge test: A critical review. *International Journal of Neuropsychopharmacology*, 1, 49–69.
- Nickel, K. M., Nickel, C., & Kaplam, C. (2005). Treatment of aggression with topiramate in male borderline patients: A double-blind, placebo-controlled study. *Biological Psychiatry*, 57, 495–499.

- Nishizawa, S., Benkelfat, C., Young, S. N., Leyton, M., Mzengeza, S., De Montigny, C., et al. (1997). Differences between males and females in rates of serotonin synthesis in human brain. *Proceedings of the National Academy of Sciences*, 94, 5308–5313.
- Olweus, D. (1979). Stability of aggressive reaction patterns in males: A review. *Psychological Bulletin*, 86, 852–875.
- Orth, U., & Wieland, E. (2006). Anger, hostility and posttraumatic stress disorder in trauma-exposed adults: A meta-analysis. *Journal of Consulting & Clinical Psychology*, 74, 698–706.
- Ouimette, P., Cronkite, R., Prins, A., & Moos, R. H. (2004). Posttraumatic stress disorder, anger and hostility, and physical health status. *Journal of Nervous and Mental Disease*, 192, 563–566.
- Paris, J., Zweig-Frank, H., Kin, N. M., Schwartz, G., Steiger, H., & Nair, N. P. (2004). Neurobiological correlates of diagnosis and underlying traits in patients with borderline personality disorder compared with normal controls. *Psychiatry Research*, 121, 239–52.
- Pasquini, M., Picardi, A., Biondi, M., Gaetano, P., & Morosini, P. (2004). Relevance of anger and irritability in outpatients with major depressive disorder. *Psychopathology*, 37, 155–160.
- Perlis, R. H., Smoller, J. W., Fava, M., Rosenbaum, J. F., Nierenberg, A. A., & Sachs, G. S. (2004). The prevalence and clinical correlates of anger attacks during depressive episodes in bipolar disorder. *Journal of Affective Disorders*, 79, 291–295.
- Pitman, R. K., & Delahanty, D. L. (2005). Conceptually driven pharmacologic approaches to acute trauma. *CNS Spectrums*, 10, 99–106.
- Pjrek, E., Winkler, D., & Kasper, S. (2005). Pharmacotherapy of seasonal affective disorder. *CNS Spectrums*, 10, 664–669.
- Placidi, G. P. A., Oquendo, M. A., Malone, K., Huang, Y. Y., Ellis, K. M., & Mann, J. J. (2001). Aggressivity, suicide attempts, and depression: Relationship to cerebrospinal fluid monoamine metabolite levels. *Biological Psychiatry*, 50, 783–791.
- Posternak, M. A., & Zimmerman, M. (2002). Anger and aggression in psychiatric outpatients. *Journal of Clinical Psychiatry*, 63, 665–672.
- Prochazka, H., & Agren, H. (2003). Self-rated aggression and cerebral monoaminergic turnover. Sex differences in patients with persistent depressive disorder. *European Archives of Psychiatry and Clinical Neuroscience*, 253, 185–192.
- Ravindran, A. V., Griffiths, J., Reali, Z., Knott, V. J., & Anisman, H. (1999). Influence of acute tryptophan depletion on mood and immune measures in healthy males. *Psychoneuroendocrinology*, 24, 99–113.
- Rinne, T., Westenberg, H. G. M., den Boer, J. A., & van den Brink, W. (2000). Serotonergic blunting to meta-chlorophenylpiperazine (m-cpp) highly correlates with sustained childhood abuse in impulsive and autoaggressive female borderline patients. *Biological Psychiatry*, 47, 548–556.
- Rinne, T., van den Brink, W., Wouters, L., & van Dyke, R. (2002). SSRI treatment of borderline personality disorder: A randomized, placebo-controlled clinical trial for female patients with borderline personality disorder. *American Journal of Psychiatry*, 159, 1048–2054.
- Roiser, J. P., McLean, A., Ogilvie, A. D., Blackwell, A. D., Bamber, D. J., Goodyer, I., et al. (2005). The subjective and cognitive effects of acute phenylalanine and tyrosine depletion in patients recovered from depression. *Neuropsychopharmacology*, 30, 775–785.
- Rosenbaum, J. F., Fava, M., Pava, J. A., McCarthy, M. K., Steingard, R. J., & Bouffides, E. (1993). Anger attacks in unipolar depression, Part 2: Neuroendocrine correlates and changes following fluoxetine treatment. *American Journal of Psychiatry*, 150, 1164–8.
- Roy, A., Adinoff, B., & Linnoila, M. (1988). Acting out hostility in normal volunteers: Negative correlation with levels of 5HIAA in cerebrospinal fluid. *Psychiatry Research*, 24, 187–194.
- Roy, A., De Jong, J., & Linnoila, M. (1989). Extraversion in pathological gamblers: Correlates with indexes of noradrenergic function. *Archives of General Psychiatry*, 46, 679–681.
- Salomon, R. M., Mazure, C. M., Delgado, P. L., Mendia, P., & Charney, D. S. (1994). Serotonin function in aggression: The effect of acute plasma tryptophan depletion in aggressive patients. *Biological Psychiatry*, 35, 570–572.
- Salzman, C., Wolfson, A. N., Schatzberg, A., Looper, J., Henke, R., Albanese, M., Schwartz, J., & Miyawaki, E. (1995). Effect of fluoxetine on anger in symptomatic volunteers with borderline personality disorder. *Journal of Clinical Psychopharmacology*, 15, 23–9.
- Schmeck, K., Sadigorshy, S., Engler, E., Demisch, L., Dierks, T., Barta, S., et al. (2002). Mood changes following acute tryptophan depletion in healthy adults. *Psychopathology*, 35, 234–240.
- Sheard, M. H. (1975). Lithium in the treatment of human aggression. *Journal of Nervous and Mental Disease*, 160, 108–118.
- Siever, L. I., Coccaro, E., & Davis, K. L. (1986). Hormone response to noradrenergic challenge. In U. Halbreich & R. Rose (Eds.), *Hormones in depression* (pp. 59–73). New York: Raven Press.

- Soloff, P. H., Cornelius, J., George, A., et al. (1993). Effects of phenelzine and haloperidol in borderline personality disorder. *Archives of General Psychiatry*, 50, 377–385.
- Soloff, P. H., George, A., Nathan, S., et al. (1989). Amitriptyline versus haloperidol in borderlines: Final outcomes and predictors of response. *Journal of Clinical Psychopharmacology*, 9, 238–246.
- Spielberger, C. D., Jacobs, G., Russell, L., & Crane, R. S. (1983). Assessment of anger: the State-Trait Anger Scale. In J. N. Butcher & C. D. Spielberger (Eds.), *Advances in personality assessment* (Vol 2, pp 161–189). Hillsdale, NJ: Lawrence Erlbaum Associates Publishers.
- Stanley, B., Molcho, A., Stanley, M., Winchel, R., Gameroff, M. J., Parsons, B., et al. (2000). Association of aggressive behaviour with altered serotonergic function inpatients who are not suicidal. *American Journal of Psychiatry*, 157, 609–614.
- Stein, D. J., Hollander, E., DeCaria, C. M., Simeon, D., Cohen, L., & Aronowitz, B. (1996). m-Chlorophenylpiperazine challenge in borderline personality disorder: Relationship of neuroendocrine response, behavioural response and clinical measures. *Biological Psychiatry*, 40, 508–513.
- Steiner, M., & Born, L. (2000). Advances in the treatment of premenstrual dysphoria. *CNS Drugs*, 13, 287–304.
- Steiner, M., & Pearlstein, T. (2000). Premenstrual dysphoria and the serotonin system: Pathophysiology and treatment. *Journal of Clinical Psychiatry*, 61(Suppl 12), 17–21.
- Tedlow, J., Leslie, V., Keefe, B. R., Alpert, J., Nierenberg, A. A., Rosenbaum, J. F., et al. (1999). Axis I and Axis II disorder comorbidity in unipolar depression with anger attacks. *Journal of Affective Disorders*, 52, 217–223.
- Trestman, R. L., Coccaro, E. F., Weston, S., Mitropoulou, V., Ramella, F., Gabriel, S., et al. (1992). Impulsivity, suicidal behavior, and major depression in the personality disorders: Differential correlates with noradrenergic and serotonergic function. *Biological psychiatry*, 31, 68A.
- Tritt, K., Nickel, C., Lahmann, C., Leiberich, P. K., Rother, W. K., Loew, T. H., et al. (2005). Lamotrigine treatment of aggression in female borderline-patients: A randomized, double-blind, placebo-controlled study. *Journal of Psychopharmacology*, 19, 287–291.
- Tse, W. S., & Bond, A. J. (2002). Serotonergic intervention affects both social dominance and affiliative behaviour. *Psychopharmacology*, 161, 324–330.
- Tse, W. S., & Bond, A. J. (2003). Reboxetine promotes social bonding in healthy volunteers. *Journal of Psychopharmacology*, 17, 65–71.
- Tucker, P., Zaninelli, R., Yehuda, R., et al. (2001). Paroxetine in the treatment of chronic posttraumatic stress disorder: results of a placebo-controlled, flexible-dosage trial. *Journal of Clinical Psychiatry*, 62, 860–868.
- Tuinier, S., Verhoeven, W. M. A., & van Praag, H. M. (1995). Cerebrospinal fluid 5-hydroxyindoleacetic acid and aggression: A critical reappraisal of the clinical data. *International Clinical Psychopharmacology*, 10, 147–156.
- van der Kolk, B. A., Dreyfuss, D., Michaels, M., et al. (1994). Fluoxetine in posttraumatic stress disorder. *Journal of Clinical Psychiatry*, 55, 517–522.
- Van Etten, M. L., & Taylor, S. (1998). Comparative efficacy of treatments for post-traumatic stress disorder: A meta-analysis. *Clinical Psychology and Psychotherapy*, 5, 126–145.
- Van Moffaert, M., & Dierick, M. (1999). Noradrenaline (norepinephrine) and depression. Role in aetiology and therapeutic implications. *CNS Drugs*, 12, 293–305.
- van Praag, H. M., Kahn, R. S., Asnis, G. M., et al. (1987). Desonologization of biological psychiatry or the specificity of 5-HT disturbances in psychiatric disorders. *Journal of Affective Disorders*, 13, 1–8.
- Velasco, C., & Bond, A. J. (1998). Personal relevance is an important dimension for visceral reactivity in emotional imagery. *Cognition and Emotion*, 12, 231–242.
- Virkkunen, M., Nuutila, A., Goodwin, F. K., & Linnoila, M. (1987). Cerebrospinal fluid monoamine metabolite levels in male arsonists. *Archives of General Psychiatry*, 44, 241–247.
- Wagstaff, A. J., Cheer, S. M., Matheson, A. J., Ormrod, D., & Goa, K. L. (2002). Paroxetine: An update of its use in Psychiatric disorders in adults. *Drugs*, 62, 655–703.
- Weltzin, T. E., Fernstrom, M. H., Fernstrom, J. D., Neuberger, S. K., & Kaye, W. H. (1995). Acute tryptophan depletion and increased food intake and irritability in bulimia nervosa. *American Journal of Psychiatry*, 152, 1668–1671.
- Wetzler, S., Kahn, R. S., Asnis, G. M., Korn, M., & van Praag, H. M. (1991). Serotonin receptor sensitivity and aggression. *Psychiatry Research*, 37, 271–279.
- Wikander, I., Sunblad, C., Andersch, V., et al. (1998). Citalopram in premenstrual disorder: Is intermittent treatment during luteal phase more effective than continuous medication throughout the menstrual cycle? *Journal of Clinical Psychopharmacology*, 18, 390–398.
- Wingrove, J., & Bond, A. J. (2005). Correlation between trait hostility and faster reading times for sentences describing angry reactions to ambiguous situations. *Cognition & Emotion*, 19, 463–472.
- Wingrove, J., Bond, A. J., & Cleare, A. J. (1999). Tryptophan enhancement/depletion and reactions to failure on a cooperative computer game. *Neuropsychopharmacology*, 21, 755–764.

- Winkler, D., Pjrek, E., Kindler, J., Heiden, A., & Kasper, S. (2006a). Anger attacks in seasonal affective disorder. *International Journal of Neuropsychopharmacology*, 9, 215–219.
- Winkler, D., Pjrek, E., Konstantinidis, A., Praschak-Reider, N., Willeit, M., Stastny, J., et al. (2006b). Validation of a simplified definition of anger attacks. *Psychotherapy and Psychosomatics*, 75, 103–106.
- Young, S. N., Smith, S. E., Pihl, R. O., & Ervin, F. R. (1985). Tryptophan depletion causes a rapid lowering of mood in normal males. *Psychopharmacology*, 87, 173–177.
- Yudofsky, S. C., Silver, J. M., Jackson, W., Endicott, J., & Williams, D. (1986). The overt aggression scale for the objective rating of verbal and physical aggression. *American Journal of Psychiatry*, 143, 35–39.
- Zanarini, M. (2004). Update on pharmacotherapy of borderline personality disorder. *Current Psychiatry Reports*, 6, 66–70.
- Zanarini, M. C., & Frankenburg, F. R. (2001). Olanzapine treatment of female borderline personality patients: A double-blind, placebo-controlled pilot study. *Journal of Clinical Psychiatry*, 62, 849–854.
- Zanarini, M. C., Frankenburg, F. R., & Parachini, E. A. (2004). A preliminary randomised trial of fluoxetine, olanzapine and the olanzapine-fluoxetine combination in women with borderline personality disorder. *Journal of Clinical Psychiatry*, 65, 903–907.
- Zuckerman, M., & Lubin, B. (1965). Educational and Industrial Testing Service. *The Multiple Affect Adjective Check List*. San Diego, CA.

Chapter 7

Somatovisceral Activation During Anger

Gerhard Stemmler

Abstract What is the physiological signature of anger? If not anger, which emotion would be more destined to turn a “cold” object perception into a Jamesian “hot” emotional encounter? Indeed, reports of bodily anger sensations are descriptions of heat and tension. However, the message from studies reporting physiological anger responses is more difficult to reconcile. The chapter discusses landmark studies on the differentiation between anger and fear. It is emphasized that their methodological characteristics are decisive for demonstrating or failing to show physiological anger specificity. A meta-analysis shows that anger provocation elicits strong changes in systolic and diastolic blood pressure, heart rate, number of skin conductance responses, and muscle activity. The pattern resembles the combined action of adrenaline and noradrenaline, accompanied by strong vagal withdrawal. It is argued that these coordinated changes have a functional value for the pursuit and finally the attainment of the goal of anger: to motivate individuals to avoid failure and pain by averting subordination under physically or socially caused harm and to gain superiority.

Anger, like all powerful emotions, has¹¹ a marked immediacy and salience in our experience. Almost instantaneously, we sense changes in our body. Something has a grip on us that we cannot control easily – then we fly into a passion (from *lat.* *pati*, to suffer). This common experience of people in all cultures and ages led, e.g., Aristotle to believe that mental phenomena were linked to organismic matter in a stepwise way: This link was presumed to be strong for emotion, less strong for thinking, and absent for the active intellect, which was thus considered “free.” Throughout Western thinking, a guiding idea has been that emotions entail irrationality, loss of free will, and animalistic drives; the core of this idea was that emotions are embodied (“psychophysiological symbolism,” Averill, 1974; Chapter 9 by J. Green et al., this book) and shaped by nature (“natural kinds,” Barrett, 2006).

One might think that it is quite easy to show that anger is associated with certain changes in our bodily state, since we so obviously experience anger’s actions in our body. But science is about questioning the obvious. We could ask, for example, what causes the reports about our sensed bodily changes during anger or what do these bodily changes look like? I start with the first question and in the main part of the chapter proceed to the second one.

G. Stemmler (✉)

Faculty of Psychology, Philipps-Universität, Marburg, Germany
e-mail: gerhardstemmler@online.de

This work has been supported by grants Ste405 of the Deutsche Forschungsgemeinschaft.

7.1 Sensed Bodily Changes

William James is considered the founder of the psychophysiological tradition in emotion research (Plutchik, 1980). He affirmed the layperson's belief that an emotion is its feeling. Somatovisceral responses follow directly upon the perception and interpretation of an emotion-eliciting stimulus. The sensation of these somatovisceral changes is a necessary condition for the formation of a feeling (for an extended Discussion, see Ellsworth, 1994; James, 1884); sensed bodily changes render the "cold" object perception into a "hot" emotional encounter. Consequently, there should be more or less strong associations between bodily responses to the emotion-arousing stimulus and their experienced sensation. Indeed, studies have shown the impact of afferent signals stemming from somatovisceral activity on brain systems and feelings (Berntson, Sarter, & Cacioppo, 2003; Critchley, Wiens, Rotshtein, Ohman, & Dolan, 2004; Pollatos, Gramann, & Schandry, 2007).

7.1.1 Reports of Bodily Sensations During Anger

Adults in various cultures report that during anger, the face turns red, the body tenses and feels hot, and breathing accelerates (see also Chapter 10 by Z. Kövesces, this volume). Janke (2002) showed that this knowledge is present by the age of 10. For example, reported increases in "feeling hot" were higher in anger than in fear, joy, and sadness; the other bodily changes sensed in anger (tension, fast breathing, rapid heart rate) were as high in fear (Rimé, Philippot, & Cisamolo, 1990). Similarly, across 2,921 respondents from 37 countries on five continents, Scherer and Wallbott (1994) found that anger was characterized by rapid heart rate (49.5% of respondents endorsed this sensation), tension (42.7%), fast breathing (37.1%), and feeling hot (31.7%). Compared to fear, anger ranked second in these bodily sensations, except for "feeling hot," which, however, was endorsed even more strongly in shame.

Three conclusions can be drawn from these results. First, with some degree of universality, people can differentiate the bodily sensations of anger from most other emotions. Second, the differentiation between sensed bodily changes during anger and fear depends on a *pattern* of changes and is difficult to make. Finally, the question remains how valid these reports of bodily sensations are when compared to actual somatovisceral changes. I will pursue this question in a later section after describing the physiological anger responses.

7.2 Physiological Anger Responses

There are hundreds of studies on the psychophysiological anger response. At the time of this writing, PsycINFO lists 603 entries for "anger and cardiovascular," 510 for "anger and blood pressure," 494 for "anger and (biology or biological)," 322 for "anger and (psychophysiology or psychophysiological)," and 1,029 for "anger and (physiology or physiological)." Many of these studies report associations of anger with clinical or subclinical states (e.g., high blood pressure, treatment programs), effects of anger on just one physiological response, or effects of just one emotion, namely anger. Such studies do not allow the drawing of conclusions about the specificity of physiological anger responses. That is why the two published meta-analyses on physiological emotion specificity identified only few qualified studies (Cacioppo, Berntson, Larsen, Poehlmann, & Ito, 2000, 18 studies comparing anger vs. other emotions published up to 1997; Stemmler, 2004, 15 studies

comparing anger vs. fear published up to 2001). Results from these meta-analyses will be presented after some landmark studies are discussed in the following section.

7.2.1 Responses in Anger Versus Control Conditions

The studies described here differ in many aspects: The kind of emotion induction, sample sizes, the timing of recording periods, baseline assessment, the assessment of components of emotion other than the physiological (e.g., emotion self-report, facial expression, voice), the statistical analysis, and whether and how the situational context was controlled for. Situational context control is necessary because every emotion induction works through a situational mediator (e.g., a film, autobiographical recall, interpersonal interaction) which leaves its own traces in physiological activity. To be sure, emotion responses should be captured free from such situational context effects. Otherwise we find in separate studies different “anger” physiologies only because the induction contexts differed.

7.2.1.1 Ax (1953)

This is *the* classical study on the psychophysiology of fear and anger; it claimed physiological specificity for these emotions. A partly identical experiment with an overlapping set of subjects was published by Schachter (1957), who gave a more complete description of the experiment than Ax did. Subjects were 43 unemployed women and men. They were asked to lie down on a bed and rest before the emotions were induced by “real-life” situations presented in a balanced order. Anger was induced by an incompetent, arrogant, and previously fired technician, who helped out “just today” (his successor was supposedly ill). Without permission of the experimenter, the technician checked the wiring, abruptly turned off the calming music played in a rest period, handled the subjects roughly and painfully, and criticized them without reason. Fear was induced by a putative life-threatening electrical short-circuit in the recording apparatus.

For statistical analysis, emotion effects were captured as difference scores between induction and prestimulus phases. During anger, heart rate, stroke volume, systolic and diastolic blood pressure, respiration rate, skin conductance level, and number of responses as well as the electromyogram (EMG) of the *m. frontalis* increased (reported are the maxima during the induction period!), whereas finger and face skin temperature fell (the minima were reported). Seven of the 14 physiological variables differentiated between the anger and fear inductions. Specific responses for anger were larger increases in diastolic blood pressure, muscle tension, and number of skin conductance responses, as well as larger decreases in heart rate minima than for fear. Ax integrated the observed physiological profile differences in the hypothesis that the physiological anger pattern resembles the effects of a mixed adrenergic–noradrenergic secretion, and the fear pattern, an adrenergic secretion.¹

¹Adrenaline stimulates alpha-adrenergic, beta1- and beta2-adrenergic receptors. Adrenergic responses are defined as reductions in finger temperature (probably because blood is redistributed from the skin to skeletal muscles), diastolic blood pressure, and total peripheral resistance and as increases in heart rate, systolic blood pressure, stroke volume, left-ventricular contractility, cardiac output, number of skin conductance responses, and respiration rate. Noradrenaline stimulates alpha- and beta1-adrenergic receptors. Noradrenergic responses are characterized by increases in systolic and diastolic blood pressure, left-ventricular contractility, number of skin conductance responses, and total peripheral resistance, as well as reductions in heart rate and finger temperature (Chessick et al., 1966; Löllgen, Meuret, Just, & Wiemers, 1985; Wenger et al., 1960). Compared to adrenaline, noradrenaline produces a lower heart and respiration rate, lower stroke volume and cardiac output, but a higher diastolic blood pressure and total peripheral resistance and a higher finger temperature.

Commendable features of Ax' experiment are the credibility, "vividness," and probably the intensity of the emotion inductions, the multivariate physiology, and the use of a non-student sample. But there are also some important limitations. First, the lack of data from other emotion components (e.g., subjective ratings of emotion) prohibits an unambiguous conclusion about the validity of the emotion inductions. In particular, it is not clear if pure anger or a fear–anger blend was induced. Second, subjects spoke and moved during the recording periods, activities that may strongly affect physiological data. Third, recording periods were overly long (7 min); increasing the likelihood of non-emotional contributions to the physiological profile. Fourth, physiological minima and maxima in long recording periods are not representative of the whole period and are less reliable than means or medians. Fifth, the calculated difference score contains both emotion and context effects. Generalizability of results can be achieved only if the effects of the non-emotional context are markedly reduced or eliminated. Finally, the statistical analysis reported was incomplete, only post hoc calculations produced the estimates of the physiological anger response noted above.

7.2.1.2 Funkenstein, King, and Drolette (1954)

This study also advanced the idea that anger is associated with a mixed adrenergic–noradrenergic physiological response (more on this below), but the unique feature of this study is the differential perspective taken. Not all subjects respond alike to the same emotion stimulus (see Chapter 15 by T. Wranik & K.R. Scherer, this volume). Consequently, subjects could be partitioned into subgroups of similar affective experience before their physiological patterns are calculated.

The authors studied 69 subjects in a frustrating number task. Subjects had to repeat six 10-digit numbers forward and backward. Then they had to solve difficult mental arithmetic tasks. Subjects were criticized when they made errors, and finally their complete failure was stated condescendingly. After that physiological data recording began. In an ensuing interview, the subjects' feelings were assessed for the mixture of anger and anxiety (anger out, anger in, anger equally out and in, equal anger and anxiety, anxiety, no emotion, miscellaneous). The anger out group ($N = 21$) showed significant percent increases from baseline to the post-provocation period in heart rate, systolic and diastolic blood pressure, and, from a probably quite unreliable ballistocardiographic recording, an increase in total peripheral resistance as well as a reduction in stroke volume and no change in cardiac output. Compared to the anxiety group ($N = 9$), the anger out group had a lower heart rate, systolic blood pressure, stroke volume, and cardiac output response, a larger response in total peripheral resistance, and an equal diastolic blood pressure response.

The strengths of this study are (1) the assessment of feeling states, (2) the use of this information to partition the sample (more on post-experimental subject selection, see Stemmler, 2003) for a potentially more concise assessment of the physiological anger response, and (3) a separation of task and recording period. Again the major problem of this study is the lack of a control condition that would permit the emotion and non-emotion effects of the induction procedure to be disentangled. A non-emotion effect in this study is the mental effort exerted during the task (for an experimental proof of such effects in a public speaking task, see Erdmann & Baumann, 1996) limiting the generalizability of the anger response data.

7.2.1.3 Levenson, Ekman, and Friesen (1990)

In this publication, the authors introduced a new emotion induction technique in a series of three experiments: The Directed Facial Action Task. Subjects were instructed to contract and hold several facial muscles. This procedure is based on the assumption ("facial feedback hypothesis") that the expression of emotions, especially in the face, would enable the corresponding affect program in the

brain and thus also its specific somatovisceral activations. Subjects followed sequential instructions to voluntarily contract sets of universally recognized facial muscle configurations of anger, disgust, fear, happiness, sadness, and surprise. Before each trial, subjects produced a standard control face that served as a baseline for the subsequent emotion face. In the first experiment, the authors varied the situational context and presented the subjects with both the Directed Facial Action Task and an imagery task, which yielded different autonomic patterns of emotion.

Levenson et al. (1990) reported means in four physiological variables across the three experiments. Compared to the control face, the anger face produced a higher heart rate, finger skin temperature, and skin conductance level. No differences were found for the muscle activity at the forearm flexor. A difference between the anger and the fear face was seen only in the higher finger skin temperature of anger. The authors interpreted the results as a clear evidence for emotion-specific autonomic activity.

The experiments of this study demonstrate the methodological advancement across the 40 years since the Ax or Funkenstein studies. There was good context control because the baseline was a control face and not just a resting period. Another positive point was the induction of no less than six “primary” emotions and the extensive use of subject information to subdivide the sample with separate specificity analyses.

7.2.1.4 Sinha, Lovallo, and Parsons (1992)

This study is an excellent example for the thoughtful application of the induction of anger via imagination. Subjects came to four separate sessions. The first session served to screen subjects (according to quality of mental imagery, alexithymia, and depression/state anxiety) and obtain personalized emotion scripts. Two sets of scripts for each of the emotions of anger, fear, joy, and sadness and for a physical action and a neutral state scene were developed by each subject, then rated for emotional content and intensity by both the subject and two independent raters. The training session allowed subjects to train imagery effective for enhanced physiological responsivity. The two experimental sessions presented each of the six scripts in randomized order, interspersed with recovery periods that lasted until a stable physiological baseline was achieved. Imagery effects were captured as imagination minus prescript differences, context control was established by defining emotion effects as emotion imagery effect minus neutral imagery effect.

The anger imagery effect comprised increases in heart rate, systolic and diastolic blood pressure, cardiac output, and total peripheral resistance, whereas left-ventricular ejection time and stroke volume decreased. Compared to fear, anger was characterized by larger increases in diastolic blood pressure and total vascular resistance.

This was a very carefully designed study. Subjects were selected to be good imaginers and they were trained. The scenes represented rather pure emotions. A representative sample of cardiovascular variables was recorded. In addition, imagination procedures have quite low non-emotional effects on somatovisceral activation, and whatever the imagination context elicited was probably effectively controlled by the neutral script.

7.2.1.5 Stemmler, Heldmann, Pauls, and Scherer (2001)

This study performed emotion inductions of anger and fear each with two different techniques (real life and imagery). An extensive set of 29 somatovisceral variables were recorded from a sample of 158 female subjects in two experimental sessions. Real-life anger was induced in three consecutive periods with intermittent recordings of physiological activation and emotion self-report. First, subjects were presented with a difficult test of general knowledge. If they did not know the answer, they

had to say loudly “I don’t know!” After the second, almost unsolvable item, subjects were asked to speak louder. Midway through, the experimenter angrily interrupted the subject saying he could not understand her. After the task subjects were informed that they had solved only one-third of the items correctly. Second, subjects performed a mental arithmetic task silently and as quickly as possible. They were interrupted twice and asked to tell the result at the moment. The experimenter commented on the poor performance and had the subject start all over again grumbling at her. Third, subjects had to solve a difficult anagram task. After 6 of 12 words, the experimenter angrily argued with the subject for moving around too much, and at the end, he accused her of noncompliance. Physiological profiles in the three induction periods were homogeneous which allowed us to use the more reliable mean of the three periods for specificity analysis. Imagery induction of anger was performed 1 week later. Subjects were asked to recall each induction period of the real-life session as vividly as possible.

Context control was instantiated by a separate control group. Control subjects were told at the beginning of the experiment that the real objective of the study was to induce anger (or fear in the respective group). To strengthen the trustworthiness of this information, the prerecorded harassments used during the later anger induction were played. Then exactly the same experiment was performed as in the anger treatment group. That is, the control subjects were exposed to exactly the same stimuli; only their interpretation of the stimuli, and the resulting emotional responses, differed. Thus, the difference between treatment and control groups should capture the emotion effects proper. All analyses were based on these differences.

The anger real-life effect comprised changes in 19 of 29 somatovisceral variables, including increases in heart rate, systolic and diastolic blood pressure, cardiac output, skin conductance level, skin temperature at the forehead, and extensor digitorum muscle activity. Compared to fear, specific responses to anger were larger total vascular resistance, skin temperature at the forehead, and extensor digitorum muscle activity. The anger imagery effect comprised 8 of 29 somatovisceral variables, among them increases in heart rate, systolic and diastolic blood pressure, number of skin conductance responses, and skin temperature at the forehead. Differences to fear imagery were seen in diastolic blood pressure.

In sum, this study pushed methodological standards even further, with the introduction of discriminant (anger vs. fear) and convergent validity (real-life vs. imagery) tests, with multiple induction periods, and an effective though costly context control strategy. Multivariate somatovisceral recordings and multivariate statistical analysis complemented each other.

The next section moves from particular landmark studies to overviews of the field.

7.2.2 Meta-analyses of Anger Effects

Psychophysiological research on emotion specificity has been analyzed in the meta-analysis of Cacioppo et al. (2000). The methodological quality of the included experimental studies was not weighted. The authors found a higher heart rate increase for anger, fear, and sadness compared to disgust and of anger compared to happiness. Compared to fear, anger responses were higher in diastolic blood pressure, number of skin conductance responses, total peripheral resistance, facial skin temperature, and finger pulse volume. Heart rate, stroke volume, and cardiac output increases were smaller in anger than in fear. The authors’ conclusion was: “In sum, the meta-analyses indicated that even a limited set of discrete emotions such as happy, sad, fear, anger, and disgust cannot be fully differentiated by visceral activity alone . . .” (p. 184). The authors reasoned that instead “. . . the negative emotions in this literature are associated with stronger ANS [autonomic nervous system] responses than are the positive emotions” (p. 184). Their Table 11.2 reveals that 10 out of 22 somatovisceral variables significantly differentiated between negative and positive emotions; this result was

based on all of the studies reviewed. The comparison between anger and fear, however, yielded eight significant variables; and these derived from only a subset of all of the studies. Thus, contrary to the authors' conclusions, it seems that their own data reveal quite a sizable amount of differentiation between anger and fear and thus make a case at least for anger and fear's somatovisceral specificity.

A meta-analysis of somatovisceral anger and fear effects was conducted by Stemmler (2004). It was based on 15 studies which reported anger and fear contrasts in at least two somatovisceral responses. Only those response variables were considered which had been used in at least three studies. Results are shown in Table 7.1.

Table 7.1 Physiological anger and fear response

Variable	<i>k</i>	<i>d</i> Anger vs. Control	<i>d</i> Fear vs. Control	<i>d</i> Anger vs. Fear
Systolic blood pressure	11	1.81**	1.67**	−0.06
Diastolic blood pressure	11	1.58**	0.93**	0.43**
Heart rate	14	1.39**	1.32**	−0.16
Number of skin conductance responses	4	1.06**	1.15**	−0.02
Muscle activity	4	1.04**	0.32*	0.37**
Skin temperature face	4	0.68**	−0.02	0.45**
Stroke volume	4	−0.63**	−0.43*	−0.12
Skin conductance level	5	0.49**	0.12	0.18
Respiration rate	7	0.47**	0.87**	−0.41**
Cardiac output	4	0.43**	0.85**	−0.41**
Skin temperature finger	8	−0.32**	−0.68**	0.18
Total peripheral resistance	3	0.32	−0.58**	0.43**

Meta-analytic results from Stemmler (2004). *k* = Number of independent studies. *d* = Effect size *d* (positive values denote higher means for anger in column 3, for fear in column 4, and for anger in column 5). By convention, an effect size of 0.20 denotes a “small,” of 0.50 a “medium,” and of 0.80 a “large” effect (Cohen & Cohen, 1983).

* $p \leq 0.05$; ** $p \leq 0.01$.

Compared to control, the anger provocation elicited strong changes in systolic and diastolic blood pressure, heart rate, number of skin conductance responses, and muscle activity. Facial skin temperature rose, as did skin conductance level, respiration rate, cardiac output, and total peripheral resistance, whereas stroke volume and finger temperature dropped. This pattern resembles the combined action of adrenaline and noradrenaline, accompanied by strong vagal withdrawal (see Footnote 1). Compared to fear, anger was characterized by a larger response in facial temperature, diastolic blood pressure, total peripheral resistance, and muscle tension. During fear, respiration rate and cardiac output were larger than during anger. Thus, 6 out of 15 variables indicated specific responses when anger was compared to fear. But there were strong responses to anger which overlapped considerably with responses to fear, such as heart rate or systolic blood pressure. I will return to this point later.

The meta-analysis also revealed that the studies were significantly heterogeneous with respect to the effect sizes just noted. Three potential moderator variables were examined for their ability to reduce this heterogeneity and to gather additional information about the conditions under which the specificity of the physiological anger response was largest. The analysis of moderator variables was restricted to data sets with at least 10 studies. This criterion left only heart rate and systolic and diastolic blood pressure as outcome variables.

The first moderator variable was the induction context (imagination; real life). Somatovisceral anger versus control effect sizes did not depend on the induction context; compared to fear, however, anger specificity in diastolic blood pressure was very large, when imagination (Cohen's effect size $d = 1.06$) was the induction method, but it was low during real-life inductions ($d = 0.18$). The second moderator variable was the design of the emotion effect (within-subjects, i.e., repeated emotion

inductions within individuals; or between-subjects, i.e., only one emotion induction per individual). In diastolic blood pressure, anger versus control effect sizes were larger when a within-subjects ($d = 1.85$) rather than a between-subjects design of the emotion effect ($d = 1.06$) was used. A within-subjects design ($d = 0.72$) was also preferable when anger specificity was probed (between-subjects design: $d = 0.14$). The third moderator was the design of the control strategy used (within-subjects comparison of emotion inductions with a rest, prestimulus, or poststimulus period; within-subjects comparison with a control condition that controls for context effects). Again in diastolic blood pressure, context control produced larger specificity effects ($d = 1.06$) than a simple rest period ($d = 0.47$). In sum, at least for diastolic blood pressure, these analyses suggest that the optimal study to demonstrate anger versus fear specificity uses imagination as the induction method, a repeated measures design to induce anger and fear in the same subject, and an effective control imagery.

7.3 Bodily Sensations and Actual Somatovisceral Responses

Earlier in this chapter I reviewed studies on bodily sensations during anger compared to fear. The result was that anger was characterized in particular by feeling hot. The meta-analysis presented above and in Table 7.1 also suggested that face temperature is a distinguishing feature of the physiological anger response. Figure 7.1 shows the standardized endorsement rates of bodily sensations for anger and fear from Scherer and Wallbott (1994) together with the standardized physiological effect sizes of Table 7.1's columns " d Anger vs. Control" and " d Fear vs. Control." Six variables matched between sensation and physiological data sets, "breathing change" – respiration rate change, "feeling cold/shivering" – finger temperature change (reversed), "feeling hot/cheeks burning" – face

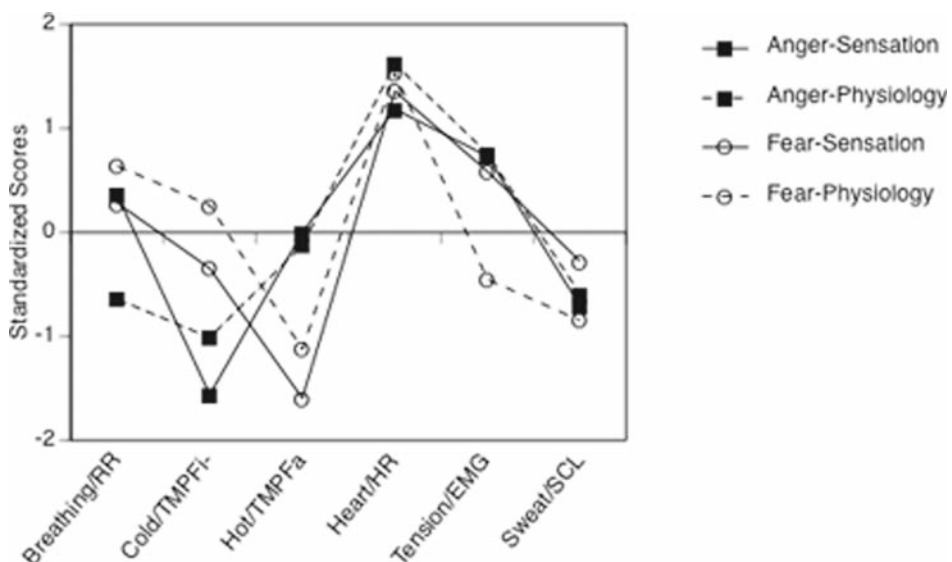


Fig. 7.1 Profiles of anger sensation, anger physiology, fear sensation, and fear physiology in matching sensation and physiology variables (z -standardized). Data for bodily sensations in anger and fear are from Scherer and Wallbott (1994), Table 8; data for physiological responses are from Stemmler's (2004) meta-analysis, see also Table 7.1 this chapter. See text for details

temperature change, “heart beating faster” – heart rate change, “muscles tensing/trembling” – muscle activity change, and “perspiring/moist hands” – skin conductance level change.

Overall, the profiles for anger and fear were quite parallel, with the notable exceptions of cold (peak for fear) and hot temperature (peak for anger) responses. Anger sensation and anger physiological profiles were highly correlated, $r = 0.85$; fear sensation and fear physiological profiles, $r = 0.78$. But between fear and anger profiles there were numerically high correlations suggesting the need to partial the bivariate correlations with respect to the remaining profiles. For example, the high correlation between anger sensation and anger physiology profiles to a marked extent could be due to the *indirect* influence of fear sensation and/or fear physiology. Partialing these two influences from the anger sensation and anger physiology correlation would provide a clearer picture of their *direct* association. Interestingly, anger sensation and anger physiology still correlated $r = 0.76$ as did fear sensation and fear physiology, $r = 0.74$, whereas all other correlations practically vanished. In particular, anger sensation correlated with fear physiology $r_{\text{partial}} = -0.02$; fear sensation correlated with anger physiology $r_{\text{partial}} = 0.29$. These purely descriptive data suggest that anger profiles of sensation and physiology have a common and specific core once the influence of fear is partialled out, and the same is true for fear. That is, on top of rather general, valenced bodily feelings (this feels good or bad) sensations seem to correspond quite well to the actual physiological changes. While the present analysis can only be suggestive, not conclusive, it is nonetheless consistent with the pan-cultural linguistic metaphors for the experience of anger based on somatovisceral physiology (Chapter 10 by A. Kövesces, this book) and justification for the inclusion of physiological imagery in anger-control psychotherapy that is the antithesis of anger-associated changes (e.g., cooling sensations in the body, Chapter 28 by E. Fernandez, this book.)

The functional relationship between physiological responses and self-reports of feeling has a long history (Cannon, 1927; James, 1884, 1894) that cannot be covered here in any detail. The debate was centered around the question whether physiological changes in emotion are *necessary* for feelings of emotion. Behind that question lurked the distinction between peripheralist versus centralist (Fehr & Stern, 1970) and – enlarged today – between biological versus cognitive explanations of emotion (Barrett, 2006). Early on, James advocated the study of spinal cord injured patients to solve this question. But the evidence from such studies is still equivocal: Somatovisceral activation seems to have at least some importance for the experience and action tendencies following emotions (Wiens, 2005). Various processes such as central sensitization, affective vulnerability, autonomic control, and autonomic representations in the brain all seem to be involved which have diverging effects on emotion outcome variables (Cobos, Sanchez, Garcia, Vera, & Vila, 2002; Nicotra, Critchley, Mathias, & Dolan, 2006).

Researchers have speculated how discrepancies between perceived/reported and objectively measured physiological changes might be explained. It could be the case that the attribution of bodily sensations to emotions is not based on the sensations per se, but on emotion schemata which might arise from individual history and general cultural influences (Rimé et al., 1990). Since cultural influences and learned stereotypes are expected to be similar for members of one culture, the ratings of study participants could be quite consistent. This was exactly what Rimé et al. (1990) found: Reports about bodily sensations during actual emotion episodes were not different from ratings of the stereotyped picture of such sensations. But, conversely, the source of these “stereotypes” could be just the common experience of prototypical bodily changes during strong emotions. Then the ratings of the stereotype would be better called “common experience” or “valid knowledge.”

Cacioppo, Berntson, and Klein (1992) introduced another theoretical account for non-veridical reports, the Somatovisceral Afference Model of Emotion (SAME), which proposes three routes along which somatovisceral activity might shape bodily sensations. According to the SAME model, an emotional stimulus is subjected to a rapid but incomplete appraisal, which can evoke bodily

responses. These responses could be emotion specific, only partially differentiated, or completely undifferentiated. The pattern of this somatovisceral activation is fed back to the brain and evaluated, just as is the emotion-eliciting stimulus.

The goal of the cognitive evaluation is to arrive at an unequivocal statement about one's emotional state; this is best achieved with the label for a discrete feeling. In the case of an emotion-specific somatovisceral pattern, a discrete feeling would derive from accurate pattern recognition. In the case of a completely undifferentiated somatovisceral afference, "one labels, interprets and identifies this state in terms of the characteristics of the precipitating situation and of one's apperceptive mass" (Schachter, 1975, p. 530; Schachter & Singer, 1962), and thus arrives at a discrete feeling. If somatovisceral activation is only partially differentiated, emotional schemata would be prompted and would lead to emotional percepts with a high degree of definition.

7.4 The Issue of Specificity

The preceding paragraphs make it obvious that somatovisceral responses during anger – even in the laboratory and under such "non-provocative" conditions as imagery – can be quite strong. But there is also some degree of overlap in the responses to anger and other emotions, especially fear, leading some authors to the conclusion that physiological emotion specificity is at best unproven and probably a myth (Barrett, 2006; Ortony & Turner, 1990). In this section, I will argue for the case of specificity, at least for a small number of basic emotions including anger.

7.4.1 *Physiological Considerations*

7.4.1.1 The Autonomic Nervous System (ANS)

Up to the present day, claims for physiological emotion specificity are countered with the argument that the sympathetic nervous system generates only an undifferentiated and diffuse innervation of its target organs. However, physiological research contradicts this notion (Jänig, 2003, 2006).

The main task of the ANS is the distribution of specific signals of the central nervous system (CNS) to the end organs in order to achieve an optimal state of homeostasis. Interactions with the external world are carried out by motor systems. The endocrine system and the ANS support the motor systems by establishing an optimal internal milieu under changing conditions and demands. Both motor and endocrine as well as autonomic homeostatic regulations are coordinated under the control of the forebrain and they are integrated with representations of the perceptual world. Various efferent signals transmitted through pre- and postganglionic neurons are functionally separate from one another and therefore allow a very precise CNS control of the target regions. The CNS signals can be modified within the autonomic ganglia, which allows for self-regulation at various system levels.

7.4.1.2 Spinal and Supraspinal Control

The spinal cord contains many autonomic reflex centers. Supraspinal centers in the lower brain stem organize the homeostatic control of the cardiovascular, respiratory, and enteric system (e.g., blood pressure control through baroreceptor reflexes). Supraspinal centers in the upper brainstem, hypothalamus, and limbic system elicit distinct autonomic response patterns which coordinate organismic adjustments across somatomotor, autonomic, and endocrine response systems (Smith, DeVito, &

Astley, 1990). Jänig (2006) describes eight response patterns which are reliably induced during specific behaviors, among them exercise, vigilance, confrontation, or flight. Still higher centers are the anterior cingulate cortex, the insula, or the orbito- and ventromedial prefrontal cortex. These and other brain regions that exert an effect on autonomic response patterns are collectively called the “Central Autonomic Network” (Thayer & Brosschot, 2005).

The conclusion from this brief review is that the brain has the capacity to elicit specific and integrated autonomic responses. Because anger – as one of the basic emotions – is a psychobiological state, it functions to

- provide perceptual, cognitive, and organismic resources for the attainment of the emotion anger’s goal (see below),
- signal conspecifics about one’s own emotional state and an increased likelihood of aggressive responding,
- enhance intraorganismic information exchange, homeostasis and coordination, and
- protect the body against adverse consequences like injury when goal attainment becomes difficult or fails.

These actions and displays initiated by anger depend on a differentiated and well-functioning ANS.

7.4.2 Physiological Maps

For more than 50 years, multichannel physiological recordings have demonstrated that somatovisceral responses are strongly influenced by situations, individual differences, and individual-specific states (from, e.g., Lacey, Bateman, & van Lehn, 1953 to Foerster, 1985). The ANS is obviously able to produce specific patterns (Fahrenberg, 1987; Stemmler & Fahrenberg, 1989). Furthermore, somatovisceral patterns are both distinct and high stable.

Figure 7.2 presents an example of situational response specificity. Each label stands for the somatovisceral profile of one experimental situation. The axes are discriminant functions representing the plane that maximizes physiological differences between situations relative to variance among subjects within situations. The data are from Stemmler (1989). The anger induction was embedded in an anagram task, similar to the one described above. The fear induction consisted of a dramatic recitation of parts of E. A. Poe’s “The fall of the house of Usher” dubbed with anxiety-provoking music (Prokofiev’s second symphony) and ended with an unanticipated sudden darkness. The physiological anger patterns were located in the lower right quadrant, and the fear patterns, in the upper left quadrant. The patterns displayed clear distinctness and stability, as did patterns during repeated tasks, such as a simple numbers task (labels N1–N4), periods before a speech (B1–B3), or periods during a speech (D1–D3).

7.4.3 The Component Model of Somatovisceral Response Organization in Anger and Fear

It does not come as a surprise that at any given moment of time several factors may contribute to the physiological response pattern. Being angrily aroused when hit by a shopping cart while standing in line will result in different somatovisceral responses than being angrily aroused while imagining the same event in a comfortable armchair. Thus, the physiological pattern at any given moment does

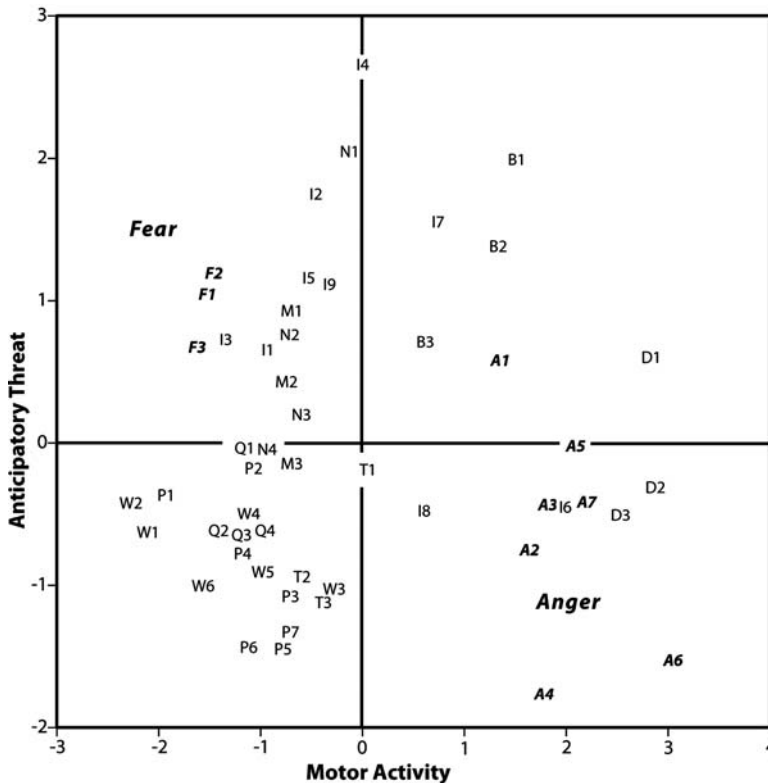


Fig. 7.2 Physiological map of 52 situations on the basis of 34 somatovisceral variables and 42 subjects (Stemmler, 1989). Bold labels denote somatovisceral patterns during induction periods for anger (A) and fear (F). A1 = Anagrams 1–15. A2 = 1st Interrupt. A3 = Anagram 16. A4 = 2nd Interrupt. A5 = Anagram 17. A6 = 3rd Interrupt. A7 = Anagrams 17–25. I6 = Instruction to wait after anger induction. F1 = Radio Play. F2 = Sudden darkness. F3 = Lights on. I = Instruction periods. M = Instruction for imagination task. P, Q = Pre- and poststimulus periods. W = Waiting periods. N = Number tasks. B, D, T = Before, during, after speech

not easily reveal which factors produced it. It could be that the differences between a physiological anger pattern and that of another emotion reflect just differences between the contexts in which the emotions were induced, and not differences between the emotions proper. Maybe physiological emotion differences do not exist after all, and all we see in the physiological recordings are emotion-unspecific effects? That is what Lang, Bradley, and Cuthbert (1990) implied when they wrote: “. . . that such physiological specificity in emotion may be tactical. That is, although specific action dispositions may be implicit in the conception of particular emotions (e.g., avoidance behavior with fear states, inhibition with sadness), they are also heavily modified by the demands of any specific context of expression” (p. 388).

The “contamination” of measures is quite a common problem in science. We may differentiate a theoretical and a pragmatic level to deal with it. Theoretically, I have proposed four different models of physiological emotion specificity, which are based on different assumptions about this confound (Stemmler, 1984, 1992b, 2003). For example, context-deviation specificity views emotion specificity as a conditional concept. An emotion “stimulus” is assumed to modify a preexisting context-bound physiological pattern. The pragmatic level, then, is to find ways to pull apart this confound. I have described experimental designs and validation strategies which constitute both necessary and sufficient conditions for claiming physiological emotion specificity (Stemmler, 1992b, 2003).

As postulated in the context-deviation model of physiological emotion specificity, various influences may impinge upon the activity of physiological variables. Just how such multiple stimuli combine in autonomic response amplitudes has been studied with two and three simultaneously acting stimuli in comparison with each stimulus administered separately (Foerster, Myrtek, & Stemmler, 1993; Myrtek & Spital, 1986). The results showed that in the majority of the physiological responses the effects of the stimuli combined synergistically and not just in an independent additive way.

In emotion research, which kinds of influences on physiological variables can be distinguished? The component model of somatovisceral response organization in anger and fear (Stemmler et al., 2001) postulates three classes of influences:

- The first component is characterized by the “non-emotional” context of an emotion induction, such as posture, ambient temperature, ongoing motor activity, or cognitive demands, which are not in the service of an emotion.
- The second component reflects a fixed (specific?) somatovisceral adaptation, which has at least two important functions: (1) the protection of the organism through autonomic reflexes and (2) the preparation of the organism for prototypical behaviors in the service of an emotion’s goal attainment. These somatovisceral emotion signatures are recognizable probably only during a rather short temporal window during and after the arousal of an emotion and before actual behavior has started. It could be that the supraspinal autonomic response generators described above produce these signatures. In the case of anger this would be the generator for the confrontation pattern.
- The third component embraces contextual resources. These are effects of organismic, behavioral, and mental demands that are necessitated by the momentary situation in the pursuit of an emotion goal. This component allows for a flexible organization of bodily resources given the momentary situational circumstances. For example, where an opportunity for “fight or flight” arises, the defense reflex is likely to be activated because it prepares the organism “to cope with an emergency and specifically to perform the extreme muscular exertion of flight or attack” (Hilton, 1982, p. 159). Depending on the context as it is physically laid out and as the individual perceives and understands it, responses elicited by this third component may produce a marked overlap of physiological responses across emotions.

What functional value can be ascribed to the somatovisceral signature of anger? Anger is a neurobehavioral system which motivates individuals to avoid failure and pain by averting subordination under physically or socially caused harm and to gain superiority. Plutchik (1980) called the prototypical adaptation pattern of anger “destruction.” Attack is a common behavioral response that requires a strong activation of sympathetic systems for its support. The behavioral response demands a persistent, isometric muscular tension. Circulatory responses under such conditions comprise an increased diastolic blood pressure and vascular resistance, which functions in opposition to the reduced effective perfusion pressures in the regions of intense muscular contraction (Buell, Alpert, & McCrory, 1986; Shanks, 1984). This means that the cardiovascular system operates to force blood to muscles whose contraction has squeezed and reduced their vascular supply. This autonomic pattern is seen already in anticipation of the handgrip task, which provokes this circulatory response (Mäntysaari, Antila, & Peltonen, 1988). The face turns red and hot, signaling an opponent the state of preparedness for attack.

Other important functional outcomes arise from interactions between the cardiovascular system and the brain. First, it is well known that rising blood pressure excites baroreceptors in the aortic arch and carotid sinus, which in turn have an inhibitory influence on pain thresholds (determined by pain ratings, see Ring, Edwards, & Kavussanu, 2008). In dangerous situations, this mechanism is certainly

advantageous. Second, behavioral and electrophysiological research shows a positive association between increased blood pressure and phasic and tonic cortical alertness, level of vigilance, and preparedness to react (Duschek, Meinhardt, & Schandry, 2006). Again, this psychophysiological mechanism is extremely important for quick and decisive action. One can imagine that when hyperaroused, this mechanism can also lead to a functional uncoupling of inhibitory, prefrontal executive control processes. Blind rage could be the consequence.

The direction of the association between blood pressure and brain activity is still unknown. On the one hand, the Central Autonomic Network exerts a steering function on the ANS, resetting, for example, set points of blood pressure regulation. On the other hand, catecholamines, especially noradrenaline, play an important role in the regulation of alertness and cortical activity. In addition, afferent sympathetic and parasympathetic fibers project into the prefrontal cortex, the insula, and the anterior cingulate (Critchley et al., 2003). Thus, there is a rich interconnection of the central and the autonomic nervous system, details of which will become more clear in the years to come.

7.4.4 The Adrenaline–Noradrenaline Hypothesis

Ax (1953) and Funkenstein, King, and Drolette (1954) suggested that the autonomic pattern during anger corresponds to the effects of a mixture of noradrenaline and adrenaline (see Footnote 1). In contrast, fear would be characterized by an autonomic pattern resembling the effects of adrenaline alone. This hypothesis was bolstered by interspecies comparisons of the relative concentrations of adrenaline and noradrenaline in the adrenal medulla. Baboons, rabbits, guinea pigs, rats, and humans possess a larger proportion of adrenaline, whereas lions, small sharks, and whales have a larger proportion of noradrenaline (Funkenstein, 1956). The observation that the noradrenaline level of ice hockey players rose markedly more than their adrenaline level, whereas the opposite was true for the coach and the substitutes, also seemed to fit the hypothesis well (Elmadjian, Hope, & Freeman, 1957). However, vigorous movement instead of anger aggression might have been responsible for these effects (see also Glass et al., 1980; Ziegler, Lake, & Kopin, 1977).

Wagner's (1989) discussion of studies on hormone secretion during emotions found them to mostly support the adrenaline–noradrenaline hypothesis. Other studies were not compatible with this interpretation, however. For example, Chessick, Bassan, and Shattan (1966) induced anger and fear, also infused their subjects with adrenaline and noradrenaline, then compared the ensuing autonomic response profiles. There were no marked similarities between emotion and catecholaminergic infusion profiles. In a review of many studies performed in her laboratory, Frankenhaeuser (1979) disputed the notion of a selective secretion of adrenaline and noradrenaline in emotional states.

The meta-analytically derived somatovisceral differences between anger and fear in Table 7.1 agree with the expected differential effects of the catecholamines in the following responses: diastolic blood pressure and total peripheral resistance (higher in anger than fear) as well as cardiac output and respiration rate (higher in fear than anger). With regard to heart rate and finger skin temperature, the direction of the empirical results does conform to expectations, but the results do not reach significance. In sum, the adrenaline–noradrenaline hypothesis of anger and fear turns out to be a good but not an exhaustive hypothesis to account for the characteristics of the respective autonomic responses.

7.4.5 Anger and Alpha-Adrenergic Activation

Another mechanism-based model of anger specificity could refer to major receptor types in the ANS. For example, alpha-adrenergic, beta-adrenergic, and cholinergic cardiovascular tone might be described as distal mechanisms and diastolic blood pressure as a proximal variable. Blockade studies suggested that diastolic blood pressure rises both with alpha-adrenergic tone (Nelson, Silke,

Hussain, Verma, & Taylor, 1984) and with loss of vagal tone (Knoebel, McHenry, Phillips, & Widlansky, 1974; Levine & Leenen, 1989). In contrast, diastolic blood pressure is not controlled by beta-adrenergic tone under resting conditions (Silke, Nelson, Ahuja, Okoli, & Taylor, 1983). Thus, diastolic blood pressure will rise both during an alpha-adrenergic state and during a state of vagal withdrawal.

If cardiovascular activity is governed to an important degree by the action of alpha-adrenergic, beta-adrenergic, and cholinergic influences, or “activation components,” then anger with its strong vascular component could be associated with an alpha-adrenergic state, probably accompanied by a marked beta-adrenergic state. In order to test this notion, Stemmler (1992a) induced anger during the third of four sessions 1 week apart and each time under a different combination of partial dual receptor blockades.² The sessions were identical with the exception of the anger induction right before a sentence completion task. Subjects were harassed for moving too much:

“What’s going on here! We’ve had it! The whole recording is totally messed up – we’ll have to junk it. Why do you think we keep asking you to sit still?! You’d think we could expect just a little more cooperation from a med-student. Now we have to go through the whole procedure again! You’ll have to come back one more time – but you know we can’t pay extra for it. – Now let’s see if at least the next task will work. – But try to control yourself a little bit this time – sit still and keep your arms as relaxed as possible and don’t talk!” (Stemmler, 1992a, p. 182)

Compared to the control group angered subjects under Placebo showed a significant increase in the activation of the beta-adrenergic component (see Fig. 7.3). Unexpectedly, the anger group under Placebo did not demonstrate an alpha-adrenergic activation (Panel a). In the “alpha-free” group,

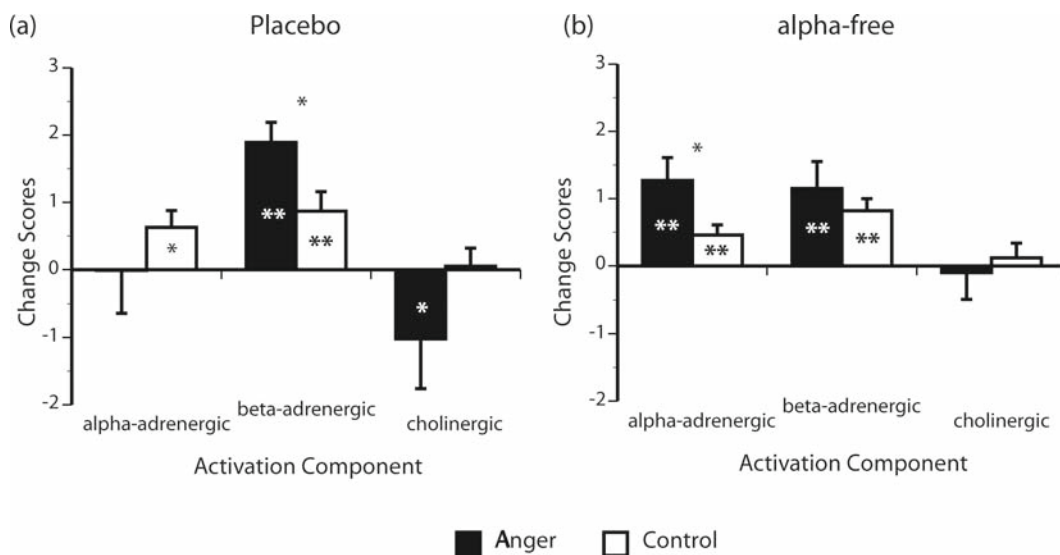


Fig. 7.3 Effects of an anger induction on three cardiovascular activation components in comparison with a control condition with identical context (Stemmler, 1992a). Panel (a) shows data of subjects under Placebo, Panel (b), under partial beta-adrenergic (60 mg Propranolol) and cholinergic blockade (1 mg Atropine sulfate; “alpha-free”). Each bar represents the mean of 12 subjects (+/- SEM). Stars inside/outside of bars denote significant differences from zero/between anger and control groups. Change scores are task minus prestimulus scores

²In a crossed carry-over design and randomized across sessions, subjects were given either Placebo, partial beta-adrenergic and cholinergic blockade (“alpha-free”), partial alpha-adrenergic and cholinergic blockade (“beta-free”), or partial alpha- and beta-adrenergic blockade (“chol-free”).

however, the beta-adrenergic activation was reduced to the level of the control group, but an alpha-adrenergic activation became apparent (Panel b). Thus, the partial beta-adrenergic receptor blockade *unmasked* the alpha-adrenergic activation, a phenomenon not unknown in the literature (Martin et al., 1974). These results suggest that anger is indeed characterized by a strong alpha-adrenergic influence, which especially in real life, but probably less so in imagery induction contexts, is masked by a strong beta-adrenergic activation.

7.4.6 The Problem of Unspecific Somatovisceral Responses

How tenable is the request that a *complete* differentiation by autonomic activity of discrete emotions is necessary to demonstrate their biological distinctness? Such a demand is not well founded. Fact is that physiological emotion responses are distinct in only a subset of recordable variables (*incomplete* differentiation). Nevertheless, the biological function of physiological emotion responses emerges only when all variables, specific and unspecific ones, are considered. For example, a heart rate increase without a strong blood pressure boost may indicate vagal withdrawal, whereas a heart rate increase together with a strong blood pressure boost may indicate a combined alpha- and beta-adrenergic activation. Without consideration of the unspecific heart rate increase the former case would no longer be interpreted as vagal withdrawal, but as a non-response.

Unspecific responses can discriminate between control and emotion induction conditions, but not between inductions of different emotions. The meta-analysis of anger and fear noted unspecific responses for heart rate, systolic blood pressure, number of unspecific skin conductance responses, and finger skin temperature. These responses can very well be a genuine part of the physiology of these emotions, for example, since they prepare for action (*fight and flight*). In order to understand the physiological “signature” and the functional meaning of an emotion, both specific and unspecific responses need scrutiny.

7.5 Conclusions

As I have tried to show, anger has a distinct somatovisceral physiology which is also sensed quite well by children and adults. At its core is an alpha-adrenergic activation, which enables continued isometric exertion of skeletal muscles. In addition, the rise in blood pressure has effects on the brain, for example, elevated pain thresholds. There are also positive associations between blood pressure and EEG arousal, which mark an increase in sustained alertness, vigilance, and preparedness to react. The prefrontal cortex, the insula, and the anterior cingulate could be the brain regions where cardiovascular arousal, regulation of pain, and cortical activation interact. These coordinated changes have a functional value for the pursuit and finally the attainment of the goal of anger: To motivate individuals to avoid failure and pain by averting subordination under physically or socially caused harm and to gain superiority. For survival and social organization of men and mice this is so important a goal that it is deeply embedded in the mammalian brains (Panksepp, 2007).

References

- Averill, J. R. (1974). An analysis of psychophysiological symbolism and its influence on theories of emotion. *Journal for the Theory of Social Behaviour*, 4, 147–190.
- Ax, A. F. (1953). The physiological differentiation between fear and anger in humans. *Psychosomatic Medicine*, 15, 433–442.

- Barrett, L. F. (2006). Are emotions natural kinds? *Perspectives on Psychological Science*, 1(1), 28–58.
- Berntson, G. G., Sarter, M., & Cacioppo, J. T. (2003). Ascending visceral regulation of cortical affective information processing. *European Journal of Neuroscience*, 18(8), 2103–2109.
- Buell, J. C., Alpert, B. S., & McCrory, W. W. (1986). Physical stressors as elicitors of cardiovascular reactivity. In K. A. Matthews, S. M. Weiss, T. Detre, T. M. Dembrowski, B. Falkner, S. B. Manuck, & R. B. Williams (Eds.), *Handbook of stress, reactivity, and cardiovascular disease* (pp. 127–144). New York: Wiley.
- Cacioppo, J. T., Berntson, G. G., & Klein, D. J. (1992). What is an emotion? The role of somatovisceral afference, with special emphasis on somatovisceral "illusions". In M. S. Clark (Ed.), *Emotion and social behavior: Review of personality and social psychology* (pp. 63–98). Newbury Park, CA: Sage.
- Cacioppo, J. T., Berntson, G. G., Larsen, J. T., Poehlmann, K. M., & Ito, T. A. (2000). The psychophysiology of emotion. In M. Lewis & J. M. Haviland-Jones (Eds.), *Handbook of emotions* (2nd ed., pp. 173–191). New York: Guilford Press.
- Cannon, W. B. (1927). The James-Lange theory of emotions: A critical examination and an alternative theory. *American Journal of Psychology*, 39, 106–124.
- Chessick, R. D., Bassan, M., & Shattan, S. (1966). A comparison of the effect of infused catecholamines and certain affect states. *American Journal of Psychiatry*, 123, 156–165.
- Cobos, P., Sanchez, M., Garcia, C., Vera, M. N., & Vila, J. (2002). Revisiting the James versus Cannon debate on emotion: Startle and autonomic modulation in patients with spinal cord injuries. *Biological Psychology*, 61, 251–269.
- Cohen, J., & Cohen, P. (1983). *Applied multiple regression/correlation analysis for the behavioral sciences* (2nd ed.). Hillsdale, NJ: Lawrence Erlbaum.
- Critchley, H. D., Mathias, C. J., Josephs, O., O'Doherty, J., Zanini, S., Dewar, B. K., et al. (2003). Human cingulate cortex and autonomic control: converging neuroimaging and clinical evidence. *Brain*, 126, 2139–2152.
- Critchley, H. D., Wiens, S., Rotshtein, P., Ohman, A., & Dolan, R. J. (2004). Neural systems supporting interoceptive awareness. *Nature Neuroscience*, 7(2), 189–195.
- Duschek, S., Meinhardt, J., & Schandry, R. (2006). Reduced cortical activity due to chronic low blood pressure: An EEG study. *Biological Psychology*, 72(3), 241–250.
- Ellsworth, P. C. (1994). William James and emotion: Is a century of fame worth a century of misunderstanding? *Psychological Review*, 101(2), 222–229.
- Elmadjian, F., Hope, J. M., & Freeman, H. (1957). Methacholine test and epinephrine and arterenol excretion. *Archives of Neurology and Psychiatry*, 77, 399–405.
- Erdmann, G., & Baumann, S. (1996). Sind psychophysiologische Veränderungen im Paradigma "Öffentliches Sprechen" Ausdruck emotionaler Belastung? *Zeitschrift für Experimentelle Psychologie*, 43(2), 224–255.
- Fahrenberg, J. (1987). Theory in psychophysiology: The multi-component analysis of psychophysiological reactivity. *Journal of Psychophysiology*, 1, 9–11.
- Fehr, F. S., & Stern, J. A. (1970). Peripheral physiological variables and emotion: The James–Lange theory revisited. *Psychological Bulletin*, 74, 411–424.
- Foerster, F. (1985). Psychophysiological response specificities: A replication over a 12-month period. *Biological Psychology*, 21, 169–182.
- Foerster, F., Myrtek, M., & Stemmler, G. (1993). Reactivity to multiple stressors: A course in synergism. *Journal of Psychophysiology*, 7(2), 115–124.
- Frankenhaeuser, M. (1979). Psychoneuroendocrine approaches to the study of emotion as related to stress and coping. In R. A. Dienstbier (Ed.), *1978 Nebraska symposium on motivation* (pp. 123–161). Lincoln, NB: University of Nebraska Press.
- Funkenstein, D. H. (1956). Nor-epinephrine-like and epinephrine-like substances in relation to human behavior. *Journal of Nervous and Mental Disease*, 124, 58–68.
- Funkenstein, D. H., King, S. H., & Drolette, M. (1954). The direction of anger during a laboratory stress-inducing situation. *Psychosomatic Medicine*, 16, 404–413.
- Glass, D. C., Krakoff, L. R., Contrada, R., Hilton, W. F., Kehoe, K., Mannucci, E. G., et al. (1980). Effect of harassment and competition upon cardiovascular and plasma catecholamine responses in Type A and Type B individuals. *Psychophysiology*, 17, 453–463.
- Hilton, S. M. (1982). The defence-arousal system and its relevance for circulatory and respiratory control. *Journal of Experimental Biology*, 100, 159–174.
- James, W. (1884). What is emotion? *Mind*, 19, 188–205.
- James, W. (1894). The physical basis of emotion. *Psychological Review*, 1, 516–529.
- Jänig, W. (2003). The autonomic nervous system and its co-ordination by the brain. In R. J. Davidson, H. H. Goldsmith, & K. R. Scherer (Eds.), *Handbook of affective science* (pp. 135–186). New York: Oxford University Press.

- Jänig, W. (2006). *The integrative action of the autonomic nervous system: Neurobiology of homeostasis*. New York: Cambridge University Press.
- Janke, B. (2002). *Entwicklung des Emotionswissens bei Kindern*. Göttingen: Hogrefe.
- Knoebel, S. B., McHenry, P. L., Phillips, J. F., & Widlansky, S. (1974). Atropine-induced cardioacceleration and myocardial blood flow in subjects with and without coronary artery disease. *American Journal of Cardiology*, 33, 327–332.
- Lacey, J. I., Bateman, D. E., & van Lehn, R. (1953). Autonomic response specificity: An experimental study. *Psychosomatic Medicine*, 15, 8–21.
- Lang, P. J., Bradley, M. M., & Cuthbert, B. N. (1990). Emotion, attention, and the startle reflex. *Psychological Review*, 97, 377–395.
- Levenson, R. W., Ekman, P., & Friesen, W. V. (1990). Voluntary facial action generates emotion-specific autonomic nervous system activity. *Psychophysiology*, 27, 363–384.
- Levine, M. A., & Leenen, F. H. (1989). Role of beta 1-receptors and vagal tone in cardiac inotropic and chronotropic responses to a beta 2-agonist in humans. *Circulation*, 79, 107–115.
- Löllgen, H., Meuret, G., Just, H., & Wiemers, K. (1985). Sympathikomimetika in der Notfall- und Intensivmedizin. *Deutsches Ärzteblatt*, 82, 1951–1955.
- Mäntysaari, M. J., Antila, K. J., & Peltonen, T. E. (1988). Circulatory effects of anticipation in a light isometric handgrip test. *Psychophysiology*, 25, 179–184.
- Martin, C. E., Shaver, J. A., Leon, D. F., Thompson, M. E., Reddy, P. S., & Leonard, J. J. (1974). Autonomic mechanisms in hemodynamic responses to isometric exercise. *Journal of Clinical Investigation*, 54, 104–115.
- Myrtek, M., & Spital, S. (1986). Psychophysiological response patterns to single, double, and triple stressors. *Psychophysiology*, 23, 663–671.
- Nelson, G. I., Silke, B., Hussain, M., Verma, S. P., & Taylor, S. H. (1984). Rest and exercise hemodynamic effects of sequential alpha-1-adrenoceptor (trimazosin) and beta-adrenoceptor (propranolol) antagonism in essential hypertension. *American Heart Journal*, 108, 124–131.
- Nicotra, A., Critchley, H. D., Mathias, C. J., & Dolan, R. J. (2006). Emotional and autonomic consequences of spinal cord injury explored using functional brain imaging. *Brain*, 129, 718–728.
- Ortony, A., & Turner, T. J. (1990). What's basic about basic emotions? *Psychological Review*, 97, 315–331.
- Panksepp, J. (2007). Neurologizing the psychology of affects: How appraisal-based constructivism and basic emotion theory can coexist. *Perspectives on Psychological Science*, 2(3), 281–296.
- Plutchik, R. (1980). *Emotion – A psychoevolutionary synthesis*. New York: Harper & Row.
- Pollatos, O., Gramann, K., & Schandry, R. (2007). Neural systems connecting interoceptive awareness and feelings. *Human Brain Mapping*, 28(1), 9–18.
- Rimé, B., Philippot, P., & Cisamolo, D. (1990). Social schemata of peripheral changes in emotion. *Journal of Personality and Social Psychology*, 59, 38–49.
- Ring, C., Edwards, L., & Kavussanu, M. (2008). Effects of isometric exercise on pain are mediated by blood pressure. *Biological Psychology*, 78(1), 123–128.
- Schachter, J. (1957). Pain, fear, and anger in hypertensives and normotensives. *Psychosomatic Medicine*, 19, 17–29.
- Schachter, S. (1975). Cognition and peripheralist – centralist controversies in motivation and emotion. In M. S. Gazzaniga & C. Blakemore (Eds.), *Handbook of psychobiology* (pp. 529–564). New York: Academic Press.
- Schachter, S., & Singer, J. E. (1962). Cognitive, social, and physiological determinants of emotional state. *Psychological Review*, 69, 379–399.
- Scherer, K. R., & Wallbott, H. G. (1994). Evidence for universality and cultural variation of differential emotion response patterning. *Journal of Personality and Social Psychology*, 66, 310–328.
- Shanks, R. G. (1984). The physiological role of alpha- and beta-adrenoceptors in the regional circulation. In W. Kobinger & R. P. Ahlquist (Eds.), *Alpha and beta adrenoceptors and the cardiovascular system* (pp. 109–123). Princeton, NJ: Excerpta Medica.
- Silke, B., Nelson, G. I., Ahuja, R. C., Okoli, R. C., & Taylor, S. H. (1983). Comparative haemodynamic dose-response effects of intravenous propranolol and pindolol in patients with coronary heart disease. *European Journal of Clinical Pharmacology*, 25, 157–165.
- Sinha, R., Lovallo, W. R., & Parsons, O. A. (1992). Cardiovascular differentiation of emotions. *Psychosomatic Medicine*, 54, 422–435.
- Smith, O. A., DeVito, J. L., & Astley, C. A. (1990). Neurons controlling cardiovascular responses to emotion are located in lateral hypothalamus-perifornical region. *American Journal of Physiology*, 259, R943–R954.
- Stemmler, G. (1984). *Psychophysiologische Emotionsmuster*. Frankfurt: Lang.
- Stemmler, G. (1989). The autonomic differentiation of emotions revisited: Convergent and discriminant validation. *Psychophysiology*, 26, 617–632.

- Stemmler, G. (1992a). *Differential psychophysiology: Persons in situations*. New York: Springer.
- Stemmler, G. (1992b). The vagueness of specificity: Models of peripheral physiological emotion specificity in emotion theories and their experimental discriminability. *Journal of Psychophysiology*, 6(1), 17–28.
- Stemmler, G. (2003). Methodological considerations in the psychophysiological study of emotion. In R. J. Davidson, H. H. Goldsmith, & K. R. Scherer (Eds.), *Handbook of affective science* (pp. 225–255). New York: Oxford University Press.
- Stemmler, G. (2004). Physiological processes during emotion. In P. Philippot & R. S. Feldman (Eds.), *The regulation of emotion* (pp. 33–70). Mahwah, NJ: Erlbaum.
- Stemmler, G., & Fahrenberg, J. (1989). Psychophysiological assessment: Conceptual, psychometric, and statistical issues. In G. Turpin (Ed.), *Handbook of clinical psychophysiology* (pp. 71–104). Chichester: Wiley.
- Stemmler, G., Heldmann, M., Pauls, C. A., & Scherer, T. (2001). Constraints for emotion specificity in fear and anger: The context counts. *Psychophysiology*, 38(2), 275–291.
- Thayer, J. F., & Brosschot, J. F. (2005). Psychosomatics and psychopathology: looking up and down from the brain. *Psychoneuroendocrinology*, 30(10), 1050–1058.
- Wagner, H. (1989). The peripheral physiological differentiation of emotions. In H. Wagner & A. Manstead (Eds.), *Handbook of social psychophysiology* (pp. 77–98). Chichester: Wiley.
- Wenger, M. A., Clemens, T. L., Darsie, M. L., Engel, B. T., Estess, F. M., & Sonnenschein, R. R. (1960). Autonomic response patterns during intravenous infusion of epinephrine and nor-epinephrine. *Psychosomatic Medicine*, 22, 294–307.
- Wiens, S. (2005). Interoception in emotional experience. *Current Opinion in Neurology*, 18, 442–447.
- Ziegler, M. G., Lake, C. R., & Kopin, I. J. (1977). The sympathetic-nervous-system defect in primary orthostatic hypotension. *New England Journal of Medicine*, 296, 293–297.

Part III
Cross Cultural Expressions and Experience
of Anger

Chapter 8

The Expression of Anger Across Cultures

David Matsumoto, Seung Hee Yoo, and Joanne Chung

Abstract In this chapter, we argue that angry facial expressions have roots in our evolutionary histories and are probably genetically coded for all humans, resulting in biologically based universality in the expression and recognition of anger. At the same time, all humans live in cultures, and cultures endorse the modification of universal angry expressions. These modifications can lead to both culturally based universality as well as cultural differences in angry expressions. We argue that one of the main functions of culture is to calibrate emotional responding to culturally relevant situations, in order to maintain social order and prevent social chaos. We also present data that suggest that cultural differences in anger expression management, via mechanisms known as display rules, are associated with anger recognition accuracy rates on the cultural level. Biologically based emotions, therefore, interact with culture to produce rich and textured behavioral repertoires driven by emotion impulses.

In this chapter, we argue that angry facial expressions have roots in our evolutionary histories and are probably genetically coded for all humans, resulting in biologically based universality in the expression and recognition of anger. We base our theoretical framework on the subset of emotions known as basic emotions (Ekman, 1999), which itself is based in Darwinian and neo-Darwinian views of emotion. Although this theoretical framework has been challenged many times over the years (Feldman Barrett, 2006; Fridlund, 1994; Russell, 1994), we believe that the basic emotions framework is strongly supported by available data (reviewed below).

At the same time, all humans live in cultures, and cultures endorse the modification of universal angry expressions. These modifications can lead to both culturally based universality as well as cultural differences in angry expressions. We argue that one of the main functions of culture is to calibrate emotional responding to culturally relevant situations, in order to maintain social order and prevent social chaos. We also present data below that suggest that cultural differences in anger expression management, via mechanisms known as display rules, are associated with anger recognition accuracy rates on the cultural level. Biologically based emotions, therefore, interact with culture to produce rich and textured behavioral repertoires driven by emotion impulses. We begin by discussing biologically based, evolutionary-rooted universality in facial expressions of anger.

D. Matsumoto (✉)
San Francisco State University, San Francisco, CA, USA
e-mail: dm@sfsu.edu

8.1 Universality in Facial Expressions of Anger

In *The Expression of the Emotions in Man and Animals*, Darwin (1872/1998) claimed that all people, regardless of race or culture, possess the ability to express some emotions in exactly the same ways, primarily through their faces. Relying on advances in photography and anatomy at the time (Duchenne de Boulogne, 1862/1990), Darwin engaged in a detailed study of the muscle actions involved in emotion and concluded that the muscle actions are universal, and their precursors can be seen in the expressive behaviors of nonhuman primates and other mammals. Darwin's theory of emotion, in fact, was a major component of his theory of evolution.

Research on the basic emotions has proven Darwin to be largely (but not entirely) correct (Ekman, 1999). Basic emotions include not only anger but also disgust, fear, joy, sadness, and surprise and are distinguished from other types of emotions by several characteristics. For instance, they are elicited automatically according to universal psychological and adaptational themes (Lazarus, 1991). One theme appraised in events eliciting anger is goal obstruction (see also Chapter 15, this volume). Basic emotions are associated with unique physiological signatures in both the autonomic and central nervous systems (Davidson, Ekman, Saron, Senulis, & Friesen, 1990; Ekman, Levenson, & Friesen, 1983; Levenson, 2003; Levenson, Ekman, & Friesen, 1990) (see also Chapter 7), and these signatures have been found in widely disparate cultural groups (Levenson, Ekman, Heider, & Friesen, 1992; Mauss, Levenson, McCarter, Wilhelm, & Gross, 2005; Tsai & Levenson, 1997). For example, anger universally produces increases in heart rate, blood pressure, and increased blood flow to the arms and hands (Levenson, 2003). Basic emotions have signals that are universally expressed and recognized (Ekman, 1993; Keltner, Matsumoto, Shiota, Frank, & O'Sullivan, in press; Matsumoto, 2001), and the expressions occur spontaneously not only in the laboratory (Ekman & Rosenberg, 2005) but also in naturalistic settings (Matsumoto & Willingham, 2006). (The absence of such naturalistic observations was a major criticism of previous studies restricted to the laboratory.) Several types of studies suggest that basic emotions have an innate, genetic basis. Congenitally blind individuals spontaneously produce the same expressions (although they have difficulty posing them voluntarily) (Matsumoto & Willingham, 2009); spontaneous expressions of kin are more similar than those of non-kin (Peleg et al., 2006); expressions of monozygotic twins are more concordant than those of dizygotics (Kendler et al., 2007); and the universal expressions have been observed in nonhuman primates as well (de Waal, 2003; Parr, Waller, & Fugate, 2005), with human anger expressions analogous to primate displays of threat and aggression.

Figure 8.1 shows three examples of the universal facial expression of anger in humans. In all of them, the brows are pulled down and together by the action of the corrugator muscles. In Fig. 8.1a, b, this action is accompanied by the raising of the upper eyelid, which produces a staring quality to the eyes. In Fig. 8.1c, the corrugator action is accompanied by a tensing of the lower eyelid. In all expressions, the lips are tightened. In Fig. 8.1a, c, the mouth is closed, as if the expresser is trying to control an impending outburst. In Fig. 8.1b, the mouth is open, as if the outburst is occurring. For comparison purposes, we include a fourth picture of a primate (rhesus monkey *Macaca mulatta*) open mouth threat face, which is used by dominant animals to keep others away (Fig. 8.1d).

Darwin's (1872/1998) principle of serviceable habits includes the claim that facial expressions are the residual actions of more complete and coordinated behavioral responses involving multiple systems – vocal, postural, gestural, skeletal muscle movements, and physiological responses. That is, emotional expressions serve intrapersonal functions (Levenson, 1999). Thus the frowning of the brow and tensing of the eyelids or raising of the upper eyelids serve to fixate attention on a possible target of attack, sharpening visual focus on it. The tightening of the lips with teeth displayed is part of an attack response, the precursor of which was biting in animals. And the increased blood flow to the hands helped prepare individuals to fight.



Fig. 8.1 Examples of universal angry expressions. Photos (a) and (b) courtesy of Paul Ekman. Photo (c) from Matsumoto and Ekman's (1988) *Japanese and Caucasian Facial Expressions of Emotion*. For more information about these and other photos and for information on the Micro-Expression Training Tool, go to www.paulekman.com. Figure (d) courtesy of Lisa Parr

Emotions and their expression also have interpersonal functions (Keltner & Haidt, 1999), and angry expressions are no exception. Bringing the brows down and together while raising the upper eyelids produces a glaring, fierce expression, which can easily evoke responses in others. Angry faces evoke fear (Dimberg & Ohman, 1996) and facilitate avoidance-related behaviors in perceivers (Marsh, Ambady, & Kleck, 2005). Infants on the visual cliff who perceive their mother's anger refuse to cross the table to their mothers, while mothers' expressions of joy facilitate the crossing (Sorce, Emde, Campos, & Klinnert, 1985). We speculate that these functions are universal because anger, in its base form, probably has the same intra- and interpersonal meaning across cultures, signaling that the individual has interpreted an event as blocking a goal and is prepared to remove it. Still, research on the interpersonal functions of facial expressions is still in its infancy, and we hope that future studies can further elucidate the intimidating effects of angry expressions in social situations.

8.2 Cultural Influences on Angry Expressions

8.2.1 A Definition of Culture and Its Functions

Although emotions like anger are grounded in biology and our evolutionary history (Chapter 21), they interact with culture to produce our varied and multifarious emotional lives. Thus it is important to gain a better understanding of culture in order to understand human emotions, and our understanding of culture is based in recognition of the complexity and diversity of human social life. Humans are members of multiple groups, each having its own purpose, hierarchy, and networking system, and we move in and out of these multiple social groups constantly, creating a continual need for social adaptation. Human social life is complex because of the incredible diversity in thoughts, feelings, and actions that people are capable of having or engaging in, and social complexity is especially true of many human societies today because of the nature of the communities within which we live. Unlike many other social animals, human communities (e.g., cities) include both kin and non-kin, who live and work together.

Along with great complexity and diversity comes the greater potential for social chaos, which can easily occur especially if individuals are not well coordinated and systematically organized. Interpersonal conflict, in fact, is inevitable, and group survival is not possible without social coordination that can reduce the potential for conflict and the possibility that when such conflicts occur they

result in irreparable damage to person, property, or relationships. Coordination requires the organization and regulation of behavior, which can be achieved by rules and norms. For example, driving without laws concerning which side of the road to drive on, how to make turns, and who has the right of way would lead to chaos on the roads. Even with those laws sometimes there is near-chaos on the roads!

Culture provides necessary coordination and organization by restricting the permissible range or diversity of behavior in social interactions. This aids individuals and groups in negotiating the complexity of human social life, allowing people to move seamlessly in and out of multiple social groups, adapting behavior and adjusting along the way. Human cultures achieve this goal by providing its members with a *unique meaning and information system that allows the group to meet basic needs of survival, coordinate social behavior to achieve a viable existence, and transmit successful social behaviors* (Matsumoto, 2007; Matsumoto & Juang, 2007).

8.2.2 *The Cultural Calibration of Emotion*

One of the ways in which cultures achieve the goal of maintaining social order is by calibrating the biologically based emotion systems of its members, because emotions are a primary source of motivation for behaviors and because behaviors need to be managed in order to maintain social order (Matsumoto, Keltner, & O'Sullivan, 2007). Culturally calibrated emotions promote the production of culturally appropriate behaviors that facilitate regularity and order, while at the same time preventing culturally inappropriate behaviors that would lead to social strife. That is, one of the primary functions of culture is to elaborate, calibrate, and coordinate the emotion system with which individuals are born with culturally available events; culturally prescribed norms, values, attitudes, and beliefs; and the cultural demands of human life. The cultural calibration of emotion is necessary for members of any culture to enact normative behaviors determined by social roles specified by culturally derived meanings in specific situational contexts. In turn, culturally calibrated behaviors that occur as a result of elicited emotions reciprocally inform culture-as-a-meaning system, reinforcing values, attitudes, and norms and in some cases over time changing them.

Because culture reflects how a group solved problems of living and because human cultures need to deal with many of the same social problems, different cultures can develop similar meaning systems for many aspects of social life. We term this culturally based universality. Each culture, for instance, needs to deal with social coordination within hierarchies. Because basic emotions are universal and have the same intra- and interpersonal functions across cultures, emotions such as anger are potentially destructive in any culture because it has the potential to disrupt social bonds, cause psychological and physical harm, and invite retaliation. For these reasons, people of all cultures are likely to minimize the expression of anger toward higher-status others (Matsumoto et al., 2006).

At the same time, many cultures develop different cultural solutions to universal problems of social organization, because they exist in different ecologies and have different resources to create and engage those solutions. Differences in the physical environment, resources available, social factors, history, and types and sizes of families and communities can all affect the cultural solutions groups create in order to survive. For instance, while the minimization of destructive emotions like anger toward higher-status others may be a pancultural universal, exactly who is a higher-status person and how those emotions are minimized may be culturally variable depending on the specific meaning system associated with that culture.

Thus, cultures can differ in the specific contents of their meaning and information systems, and they can produce both similarities and differences across groups. This suggests that some aspects

of anger expressions are culturally influenced but still universal and that other aspects of anger expressions are culture-specific.

8.2.3 Cultural Calibration via Social Roles and Norms for Emotional Expression – Cultural Display Rules

Cultures calibrate the expressive and behavioral components of emotion via social roles and norms known as display rules. These are rules learned early in life that dictate the management and possible modification of an emotional response depending on social circumstances (Ekman & Friesen, 1969). They are part of the normative behaviors cultures use to produce social roles appropriate for specific situational contexts in order to preserve social order. Display rules serve a vitally important function in culture by helping to regulate emotional behaviors related to social roles and their scripts, enabling individuals to enact their social roles, which aids within-group social coordination and ultimately group and individual survival. (See Chapter 21 for an alternative explanation in terms of the adaptive value of cultural rules.)





Ekman and Friesen (1969) noted that individuals can manage their emotional expressions in a number of ways, including showing more (amplification) or less (deamplification) of the emotion they feel, showing nothing (neutralization), showing it with some other emotion (qualification), or concealing it and showing something else altogether (masking). Individuals can also show the emotion as they feel it (expression). Table 8.1 shows some examples of how these mechanisms may operate with angry expressions. These expressions were posed, but all include components of the prototypical angry expression. The top photo depicts either extreme anger (as in a rage) or an amplified emotion. The second depicts a strong angry expression by itself. The third depicts anger in the tightened lips, but it is accompanied with a smile, which qualifies the message of the anger, adding comment to the emotion, such as “I gotcha!” And the fourth expression depicts a very subtle, almost neutralized version of anger, with only a slight tightening in the lips.

Recently we obtained judgment data on these, and eight other variants of the angry expression, from approximately 50,000 individuals around the world (Matsumoto, Ekman, Witte, & Pargas, 2006). Our preliminary analyses provided glimpses as to how people can misjudge emotional expressions because of their management. For example, as you can see from the right column of Table 8.1, the vast majority of observers judged the first expression as angry; this percentage declined in the second, third, and fourth expressions. In fact, only a very small percentage of individuals were able to detect the clues to anger in the fourth expression, despite the fact that the component of the angry expression (tightened lips) is clearly present. Of course, we realize that in real life individuals receive many other cues with which to make judgments of emotion subtleties, including vocal, gestural, and contextual cues. Regardless, these data highlight how the appearance changes to angry expressions due to display rules, especially for neutralization, qualification, or masking, can alter the judgments of what emotion the expresser is feeling, sometimes dramatically.

8.2.4 Universality in Display Rules of Anger

As mentioned above, cultures can produce universality in emotional responses. For example, one important distinction that individuals in all societies make is that of ingroups and outgroups (Brewer & Kramer, 1985; Messick & Mackie, 1989; Tajfel, 1982). Self-ingroup relationships are characterized by a previous history of shared experiences and an anticipated future and produce a sense of intimacy, familiarity, and trust; self-outgroup relationships lack these qualities.

Table 8.1 Percentage of observers judging each of these expressions as anger

Expression	Judges	% Anger Judgments
	Males	69.6
	Females	74.2
	All	72.5
	Males	55.9
	Females	59.6
	All	58.2
	Males	33.8
	Females	37.0
	All	35.8
	Males	6.0
	Females	7.2
	All	6.8

Self-ingroup relationships, therefore, should be associated with norms for greater emotion expressivity because there is less anxiety resulting from the ambiguity or uncertainty of the meaning of emotional expression to the self or the relationship. Self-outgroup relationships, however, should be associated with norms for relatively less expression precisely because of the greater ambiguity and uncertainty associated with these relationships. Because these qualities characterize ingroups and outgroups panculturally, one could expect that this difference in display rules will be universal.

Indeed, this is what has been found in a 32-country survey of cultural display rules (Matsumoto et al., 2008). Participants in these countries completed a comprehensive survey of what they should do if they felt each of the seven emotions, including anger, in 42 different contexts, including with friends, acquaintances, professors, and family members. When responding, participants were able to select one of the six behavioral responses that corresponded to Ekman and Friesen's (1969) theoretical framework: amplification, expression, deamplification, neutralization, qualification, and masking. After cross-cultural equivalence was established in the measurement, the findings indicated that participants in all countries endorsed expressions of anger (and all other basic emotions) more with ingroups than with outgroups. This finding likely occurred because of the safety the familiarity the ingroup provides to the individual in relation to emotional expression. Expressing anger to strangers – a typical outgroup – may evoke retaliation or worse.

Also, participants universally endorsed more positive emotions when with family and close friends, but more modification of negative emotions, including anger, with strangers (Matsumoto et al., 2006). These display rules correspond to what has been found in studies of actual spontaneous expressive behavior (Buck, Losow, Murphy, & Costanzo, 1992; Ekman, 1972; Fridlund, 1991; Fridlund, Kenworthy, & Jaffey, 1992; Matsumoto & Kupperbusch, 2001; Wagner & Smith, 1991) and are probably produced by cross-cultural similarities in the meaning of social contexts and social roles (Matsumoto, 2007). Close friends, for instance, probably have similar psychological meaning across cultures and are associated with similar social roles (e.g., “to be a good friend”). Similarities in social roles, therefore, produce similarities in norms concerning expressive displays.

8.2.5 Cultural Differences in Display Rules of Anger

Cultures can also differ in their display rules regarding angry expressions. In the 32-country study cited immediately above (Matsumoto et al., 2008), we examined the relationship between display rules and the cultural dimension known as individualism vs. collectivism. Four attributes define this dimension (Triandis, 1995): self, goals, relationship, and determinants of behavior. Individualistic cultures foster the development of independent construals of self (Markus & Kitayama, 1991), favor personal goals over ingroup goals (Yamaguchi, 1994), encourage rationality and interpersonal exchange (Kim, Triandis, Kagitcibasi, Choi, & Yoon, 1994), and place more importance on attitudes as relatively important determinants of behavior. Collectivistic cultures foster interdependent selves and ingroup goals, encourage relatedness and communal relationships, and place relatively more importance on norms as determinants of behavior.

There was no difference in overall endorsement of angry expressions between individualistic and collectivistic cultures. But cultural differences existed when examined separately by context. Individualistic cultures were associated with greater endorsement of angry expressions toward ingroups compared to outgroups; collectivistic cultures, however, were associated with greater endorsement of angry expressions toward outgroups than ingroups. These differences likely occurred because cultures ascribe different meanings to self-ingroup and outgroup relationships (Triandis, Bontempo, Villareal, Asai, & Lucca, 1988. Members of individualistic cultures have more ingroups, and they are attached less to any single ingroup; members of collectivistic cultures belong to fewer ingroups, and their commitment to ingroups is greater than individualistic cultures (Hui, Triandis, & Yee, 1991; Pearson & Stephan, 1988; Wheeler, Reis, & Bond, 1989). Collectivistic cultures foster a greater degree of conformity within their ingroups, and sanctions exist for nonconformity (Bond & Smith, 1996). A high degree of conformity insures that individuals are identified and bonded with their ingroups, allowing groups to function and for their needs to supersede individual ones. Subjugating personal goals in favor of the group is a primary feature of collectivism, while facility in interacting with strangers is a primary feature of individualism (Oyserman, Coon, & Kimmelmeier, 2002). Thus, collectivistic cultures foster emotional displays toward ingroups that maintain and facilitate group cohesion, harmony, or cooperation to a greater degree than individualistic cultures (Matsumoto, 1991). Because anger can threaten ingroup cohesion, collectivistic cultures are associated with norms for relatively less expression of anger toward ingroups, whereas individualistic cultures are associated with norms for relatively greater angry expressions. The opposite was true for self-outgroup relationships. Here, individualistic cultures are associated with norms for relatively less anger expression toward outgroups, because doing so minimizes the differences between ingroups and outgroups and treats outgroup members like ingroup members in a relatively more

egalitarian fashion. But, collectivistic cultures are associated with relatively more anger toward outgroups, because doing so enables greater distancing between ingroups and outgroups and reinforces ingroup identity, a feature of collectivistic cultures.

There are also cultural differences in the specific ways in which angry expressions should be managed. For example, we computed the total number of times each participant endorsed each of the specific response alternatives in relation to when they were angry in the 32-country display rule data set described above and then correlated country means on the response alternatives with the five Hofstede (2001) and seven Schwartz (2004) cultural value scales. The Results were fairly clear (Table 8.2). Cultures that valued greater power distance, embeddedness, hierarchy, and long-term orientation endorsed more neutralization and masking of anger. Cultures high on individualism, affective autonomy, and egalitarianism, however, endorsed more expression and deamplification of anger. That is, embedded, hierarchical, and more collectivistic cultures encourage regulating angry expressions by neutralizing or concealing the anger; individualistic, autonomous, and egalitarian cultures encourage regulating their angry expressions by toning it down, but not eliminating it or misdirecting others by showing something else. Coupled with the judgment data described above, these cultural differences in preferred modes of angry expression regulation provide strong implications for misunderstandings in intercultural interactions.

Table 8.2 Correlations between Hofstede (2001) and Schwartz (2004) cultural values and country means on overall display rules for anger

Cultural value	Amplification	Expression	Deamplification	Neutralization	Qualification	Masking
Power distance	0.455 ^b	−0.082	−0.795 ^a	0.551 ^a	0.184	0.438 ^b
Long term-short term orientation	0.340	−0.669 ^a	−0.347	0.597 ^a	0.236	0.662 ^a
Embeddedness	0.357	−0.580 ^a	−0.643 ^a	0.696 ^a	0.351	0.648 ^a
Hierarchy	0.251	−0.723 ^a	−0.517 ^b	0.628 ^a	0.549 ^b	0.713 ^a
Individualism-collectivism	−0.373 ^b	0.291	0.664 ^a	−0.549 ^a	−0.420 ^b	−0.476 ^b
Affective autonomy	−0.487 ^b	0.490 ^b	0.703 ^a	−0.730 ^a	−0.277	−0.544 ^b
Egalitarianism	−0.243	0.748 ^a	0.584 ^a	−0.695 ^a	−0.574 ^b	−0.768 ^a
Uncertainty avoidance	−0.108	0.387 ^b	−0.161	0.149	−0.171	−0.343 ^b
Masculinity-femininity	0.076	−0.323 ^b	−0.079	0.260	0.090	0.127
Intellectual autonomy	−0.083	0.139	0.387	−0.307	−0.084	−0.374
Mastery	0.115	−0.365	−0.511 ^b	0.523 ^b	0.381	0.467 ^b
Harmony	0.001	0.270	0.177	−0.202	−0.140	−0.362

^a Correlation is significant at the 0.01 level (2-tailed).

^b Correlation is significant at the 0.05 level (2-tailed).

8.2.6 Cultural Differences in Recognizing Angry Expressions

In addition to providing cultural display rules for the management of angry expressions, cultures may also influence the perception of angry expressions (Matsumoto, 1989). And in fact, this is what has been found. Elfenbein and Ambady (2003a) meta-analyzed data from three cross-cultural

judgment studies and computed country-level correlations between Hofstede's (2001) cultural value data and the average percent recognition accuracy rates for the emotions tested. Countries high on individualism and low on power distance had significantly higher recognition accuracy rates for anger, which complements the findings reported above concerning the relationship between these cultural dimensions and expressive display rules for anger.

We took these findings a step further and gathered country-level anger recognition accuracy data for each of the countries for which we had display rule data from the 32-country data set described above. This resulted in the accumulation of data from 15 countries reported in 17 published studies (Biehl et al., 1997; Boucher & Carlson, 1980; Ducci, Arcuri, W/Georgis, & Sineshaw, 1982; Ekman, Friesen, & Ellsworth, 1972; Ekman et al., 1987; Ekman, Sorenson, & Friesen, 1969; Elfenbein & Ambady, 2003b; Elfenbein, Beaupré, Levesque, & Hess, 2007; Haidt & Keltner, 1999; Izard, 1971; Lee, Chiu, & Chan, 2005; Matsumoto, 1992; Matsumoto & Ekman, 1989; McAndrew, 1986; Niit & Valsiner, 1977; Russell, Suzuki, & Ishida, 1993; Tcherkassof & de Suremain, 2005). When multiple sources of data were available from the same country, the data were averaged across samples for that country to provide a single score in the analysis. We then computed country-level correlations between the mean anger recognition accuracy rates and the display rule expressive modes. Masking of anger was significantly negatively correlated with anger recognition rates, $r(15) = -0.51, p < 0.05$, indicating countries that endorsed the masking of anger more were less accurate in recognizing angry expressions. This finding provides empirical support for the notion that a culture's rules of expression (display rules) and perception (recognition rates) of emotion are linked (Matsumoto, 1989; Matsumoto & Ekman, 1989) and provides a basis by which cultural differences in mean recognition accuracy levels can be interpreted.

8.2.7 The Influence of Culture on Anger-Related Behaviors

As mentioned above, one of the primary functions of emotion is to prime individuals for behavior. Lowered brows and glaring eyes in angry facial expressions, for instance, aid individuals in focusing concentration on the anger-eliciting object. Pursued and tightened lips prepare the individual to bite. The bared teeth and glaring eyes are an important social signal to others. The increased heart rate and respiration increases energy for battle, while increased blood flow to the hands prepares for fighting. All of these responses are part of a universal, coordinated package of events that prepares individuals for aggressive behavior.

Fortunately, however, anger does not always result in aggression. While emotion may provide the impulse for behavior, whether or not individuals act upon those impulses is dependent on a host of factors, including the intensity of the aroused emotion, individual differences and histories, and culture. Culture provides the normative framework that describes the range of acceptable behaviors, given the social circumstances, and humans have the cognitive abilities with which to learn and engage these norms in making decisions concerning situation-appropriate behaviors when anger is elicited. When emotions are elicited, therefore, culture calibrates the primed individual to the behavioral repertoires available and necessary in that culture as identified by the social roles, norms, and expectations in order to serve as motivators for desired behaviors. Thus we view behaviors as occurring in specific situational contexts as role performances (ala Goffman, 1959) and individuals as actors playing roles as defined by culture (Matsumoto & Wilson, 2008).

Unfortunately, there is paucity of cross-cultural research examining actual behaviors in general and in relation to elicited emotions such as anger. Thus, the theoretical framework we have presented in this section must remain speculative until future research can substantiate and/or revise the framework. Such studies are sure to provide additional insights into the complex interplay between

biologically based emotions such as anger with culturally based scripts in producing behavior role performances.

8.3 Conclusion

In this chapter, we have discussed how the expression and perception of anger is at the same time universal and biologically based but also influenced by culture. Cultures endorse the modification of universal angry expressions in many ways. All of this is done to ensure culturally appropriate responding so as to maintain social order and thereby prevent social chaos. Cultural influences on expression are also associated with cultural differences in perceptions of angry expressions. Still, future research is necessary to examine the exact ways in which angry expressions are modified behaviorally in real-life contexts across cultures, and how these modifications are empirically linked to the active cultural ingredients that produce them in the first place. Such studies are difficult and time consuming; measuring behavior in equivalent setting across cultures is not an easy thing to do and neither is identifying and measuring the active cultural ingredients and linking them to the behaviors, which is what needs to occur (Matsumoto & Yoo, 2006).

References

- Biehl, M., Matsumoto, D., Ekman, P., Hearn, V., Heider, K., Kudoh, T., et al. (1997). Matsumoto and Ekman's Japanese and Caucasian facial expressions of emotion (JACFEE): Reliability data and cross-national differences. *Journal of Nonverbal Behavior*, 21, 3–21.
- Bond, R., & Smith, P. B. (1996). Culture and conformity: A meta-analysis of studies using Asch's (1952b, 1956) line judgment task. *Psychological Bulletin*, 119, 111–137.
- Boucher, J. D., & Carlson, G. E. (1980). Recognition of facial expression in three cultures. *Journal of Cross-Cultural Psychology*, 11, 263–280.
- Brewer, M. B., & Kramer, R. M. (1985). The psychology of intergroup attitudes and behavior. *Annual Review of Psychology*, 36, 219–243.
- Buck, R. W., Losow, J. I., Murphy, M. M., & Costanzo, P. (1992). Social facilitation and inhibition of emotional expression and communication. *Journal of Personality & Social Psychology*, 63(6), 962–968.
- Darwin, C. (1872/1998). *The expression of emotion in man and animals*. New York: Oxford University Press.
- Davidson, R. J., Ekman, P., Saron, C. D., Senulis, J. A., Friesen, W. V. (1990). Approach-withdrawal and cerebral asymmetry: Emotional expression and brain physiology: I. *Journal of Personality & Social Psychology*, 58(2), 330–341.
- de Waal, F. B. M. (2003). Darwin's legacy and the study of primate visual communication. In P. Ekman, J. Campos, R. J. Davidson, & F. B. M. De Waal (Eds.), *Emotions inside out: 130 years after Darwin's The expression of emotion in man and animals* (pp. 7–31). New York: New York Academy of Sciences.
- Dimberg, U., & Ohman, A. (1996). Behold the wrath: Psychophysiological responses to facial stimuli. *Motivation & Emotion*, 20(2), 149–182.
- Ducci, L., Arcuri, L., W/Georgis, T., & Sineshaw, T. (1982). Emotion recognition in Ethiopia: The effect of similarity with Western culture on accuracy of recognition. *Journal of Cross-Cultural Psychology*, 13(3), 340–351.
- Duchenne de Boulogne, G. B. (1862/1990). *The mechanism of human facial expression*. New York: Cambridge University Press.
- Ekman, P. (1972). Universal and cultural differences in facial expression of emotion. In J. R. Cole (Ed.), *Nebraska symposium on motivation*, 1971 (pp. 207–283). Lincoln, NE: Nebraska University Press.
- Ekman, P. (1993). Facial expression and emotion. *American Psychologist*, 48(4), 384–392.
- Ekman, P. (1999). Basic emotions. In T. Dalgleish, & T. Power (Eds.), *The handbook of cognition and emotion* (pp. 45–60). Sussex, UK: John Wiley and Sons, Ltd.
- Ekman, P., & Friesen, W. V. (1969). The repertoire of nonverbal behavior: Categories, origins, usage, and coding. *Semiotica*, 1, 49–98.

- Ekman, P., Friesen, W. V., & Ellsworth, P. (1972). *Emotion in the human face: Guide-lines for research and an integration of findings*. New York: Pergamon Press.
- Ekman, P., Friesen, W. V., O'Sullivan, M., Chan, A., Diacoyanni-Tarlatzis, I., Heider, K., et al. (1987). Universals and cultural differences in the judgments of facial expressions of emotion. *Journal of Personality & Social Psychology*, 53(4), 712–717.
- Ekman, P., Levenson, R. W., & Friesen, W. V. (1983). Autonomic nervous system activity distinguishes among emotions. *Science*, 221(4616), 1208–1210.
- Ekman, P., & Rosenberg, E. L. (Eds.). (2005). *What the face reveals: Basic and applied studies of spontaneous expression using the Facial Action Coding System (FACS)* (2nd ed.). New York: Oxford University Press.
- Ekman, P., Sorenson, E. R., & Friesen, W. V. (1969). Pancultural elements in facial displays of emotion. *Science*, 164(3875), 86–88.
- Elfenbein, H. A., & Ambady, N. (2003a). Cultural similarity's consequences: A distance perspective on cross-cultural differences in emotion recognition. *Journal of Cross-Cultural Psychology*, 34(1), 92–110.
- Elfenbein, H. A., & Ambady, N. (2003b). When familiarity breeds accuracy: Cultural exposure and facial emotion recognition. *Journal of Personality and Social Psychology*, 85(2), 276–290.
- Elfenbein, H. A., Beaupré, M. G., Levesque, M., & Hess, U. (2007). Toward a dialect theory: Cultural differences in the expression and recognition of posed facial expressions. *Emotion*, 7(1), 131–146.
- Feldman Barrett, L. (2006). Are emotions natural kinds? *Perspectives on Psychological Science*, 1(1), 28–58.
- Fridlund, A. (1991). Sociality of solitary smiling: Potentiation by an implicit audience. *Journal of Personality and Social Psychology*, 60(2), 229–240.
- Fridlund, A. (1994). *Human facial expression: An evolutionary view*. San Diego, CA: Academic Press.
- Fridlund, A., Kenworthy, K. G., & Jaffey, A. K. (1992). Audience effects in affective imagery: Replication and extension to dysphoric imagery. *Journal of Nonverbal Behavior*, 16(3), 191–212.
- Goffman, E. (1959). *The presentation of self in everyday life*. Oxford, England: Doubleday.
- Haidt, J., & Keltner, D. (1999). Culture and facial expression: Open-ended methods find more expressions and a gradient of recognition. *Cognition & Emotion*, 13(3), 225–266.
- Hofstede, G. H. (2001). *Culture's consequences: Comparing values, behaviors, institutions and organizations across nations* (2nd ed.). Thousand Oaks, CA: Sage Publications.
- Hui, C. H., Triandis, H. C., & Yee, C. (1991). Cultural differences in reward allocation: Is collectivism the explanation? *British Journal of Social Psychology*, 30, 145–157.
- Izard, C. E. (1971). *The face of emotion*. East Norwalk, CT: Appleton-Century-Crofts.
- Keltner, D., & Haidt, J. (1999). Social functions of emotion at four levels of analysis. *Cognition and Emotion*, 13(5), 505–521.
- Kendler, K. S., Halberstadt, L. J., Butera, F., Myers, J., Bouchard, T. J., & Ekman, P. (2007). *The similarity of facial expressions in response to emotion-inducing films in reared apart twins*. *Psychological Medicine*, 38(10), 1475–1483.
- Kim, U., Triandis, H. C., Kagitcibasi, C., Choi, S. C., & Yoon, G. (1994). Introduction. In U. Kim et al. (Eds.), *Individualism and collectivism: Theory, method and applications* (pp. 1–19). Thousand Oaks, CA: Sage.
- Lazarus, R. (1991). *Emotion and adaptation*. New York: Oxford University Press.
- Lee, S., Chiu, C.-Y., & Chan, T.-K. (2005). Some boundary conditions of the expressor culture effect in emotion recognition: Evidence from Hong Kong Chinese perceivers. *Asian Journal of Social Psychology*, 8(3), 224–243.
- Levenson, R. W. (1999). The intrapersonal functions of emotion. *Cognition and Emotion*, 13(5), 481–504.
- Levenson, R. W. (2003a). Autonomic specificity and emotion. In R. J. Davidson, K. Scherer, & H. H. Goldsmith (Eds.), *Handbook of affective sciences* (pp. 212–224). New York: Oxford University Press.
- Levenson, R. W. (2003b). Blood, sweat, and fears: The autonomic architecture of emotion. In P. Ekman, J. Campos, R. J. Davidson, & F. B. M. De Waal (Eds.), *Emotions inside out: 130 years after Darwin's "The expression of the emotions in man and animals"* (Vol. 1000, pp. 348–366). New York: New York Academy of Sciences.
- Levenson, R. W., Ekman, P., & Friesen, W. V. (1990). Voluntary facial action generates emotion-specific autonomic nervous system activity. *Psychophysiology*, 27(4), 363–384.
- Levenson, R. W., Ekman, P., Heider, K., & Friesen, W. V. (1992). Emotion and autonomic nervous system activity in the Minangkabau of West Sumatra. *Journal of Personality & Social Psychology*, 62(6), 972–988.
- Markus, H. R., & Kitayama, S. (1991). Culture and the self: Implications for cognition, emotion, and motivation. *Psychological Review*, 98, 224–253.
- Marsh, A. A., Ambady, N., & Kleck, R. E. (2005). The effects of fear and anger facial expressions on approach- and avoidance-related behaviors. *Emotion*, 5(1), 119–124.
- Matsumoto, D. (1989). Cultural influences on the perception of emotion. *Journal of Cross-Cultural Psychology*, 20(1), 92–105.

- Matsumoto, D. (1991). Cultural influences on facial expressions of emotion. *Southern Communication Journal*, 56, 128–137.
- Matsumoto, D. (1992). American-Japanese cultural differences in the recognition of universal facial expressions. *Journal of Cross-Cultural Psychology*, 23(1), 72–84.
- Matsumoto, D. (2001). Culture and emotion. In D. Matsumoto (Ed.), *The handbook of culture and psychology* (pp. 171–194). New York: Oxford University Press.
- Matsumoto, D. (2007). Culture, context, and behavior. *Journal of Personality*, 75(6), 1285–1320.
- Matsumoto, D., & Ekman, P. (1988). Japanese and Caucasian Facial Expressions of Emotion and Neutral Faces (JACFEE and JACNeuF). Available from www.paulekman.com.
- Matsumoto, D., & Ekman, P. (1989). American-Japanese cultural differences in intensity ratings of facial expressions of emotion. *Motivation & Emotion*, 13(2), 143–157.
- Matsumoto, D., Ekman, P., Witte, J., & Pargas, R. (2006). Anger, angrier, angriest: Can you tell? Publication: <http://magma.nationalgeographic.com/ngm/survey2005/>
- Matsumoto, D., & Juang, L. (2007). *Culture and psychology* (4th ed.). Belmont, CA: Wadsworth.
- Matsumoto, D., Keltner, D., & O'Sullivan, M. (2007). *Culture and emotion*. Manuscript currently submitted for publication.
- Matsumoto, D., & Kupperbusch, C. (2001). Idiocentric and allocentric differences in emotional expression and experience. *Asian Journal of Social Psychology*, 4, 113–131.
- Matsumoto, D., & Willingham, B. (2006). The thrill of victory and the agony of defeat: Spontaneous expressions of medal winners at the 2004 Athens Olympic Games. *Journal of Personality and Social Psychology*, 91(3), 568–581.
- Matsumoto, D., & Willingham, B. (2009). *Spontaneous facial expressions of emotion of blind individuals*. Manuscript currently submitted for publication.
- Matsumoto, D., & Wilson, J. (2008). Culture, emotion, and motivation. In R. M. Sorrentino, & S. Yamaguchi (Eds.), *Handbook of motivation and cognition across cultures*. New York: Elsevier.
- Matsumoto, D., & Yoo, S. H. (2006). Toward a new generation of cross-cultural research. *Perspectives on Psychological Science*, 1(3), 234–250.
- Matsumoto, D., Yoo, S. H., Alexandre, J., Altarriba, J., Anguas-Wong, A. M., Arriola, M., et al. (2006). *Universal effects of contexts on display rules for emotional behaviors*. Manuscript currently submitted for publication.
- Matsumoto, D., Yoo, S. H., Fontaine, J. R. J., Anguas-Wong, A. M., Arriola, M., Ataca, B., et al. (2008). Mapping expressive differences around the world: The relationship between emotional display rules and Individualism v. Collectivism. *Journal of Cross-Cultural Psychology*, 39(1), 55–74.
- Matsumoto, D., Keltner, D., Shiota, M. N., O'Sullivan, M., & Frank, M. (2008). Facial expressions of emotion. In M. Lewis, J. M. Haviland-Jones, & L. F. Barrett (Eds.), *Handbook of emotions* (3rd ed., pp. 211–234). New York: Guilford Press.
- Mauss, I. B., Levenson, R. W., McCarter, L., Wilhelm, F. L., & Gross, J. J. (2005). The tie that binds? Coherence among emotion experience, behavior, and physiology. *Emotion*, 5(2), 175–190.
- McAndrew, F. T. (1986). A cross-cultural study of recognition thresholds for facial expressions of emotion. *Journal of Cross-Cultural Psychology*, 17(2), 211–224.
- Messick, D. M., & Mackie, D. M. (1989). Intergroup relations. *Annual Review of Psychology*, 40, 45–81.
- Niit, T., & Valsiner, J. (1977). Recognition of facial expressions: An experimental investigation of Ekman's model. *Tartu Riikliku Ülikooli Toimetised: Trudy po Psikhologii*, 429, 85–107.
- Oyserman, D., Coon, H. M., & Kemmelmeier, M. (2002). Rethinking individualism and collectivism: Evaluation of theoretical assumptions and meta-analyses. *Psychological Bulletin*, 128(1), 3–72.
- Parr, L. A., Waller, B. M., & Fugate, J. (2005). Emotional communication in primates: Implications for neurobiology. *Current Opinion in Neurobiology*, 15(6), 716–720.
- Pearson, V. M. S., & Stephan, W. G. (1988). Preferences for styles of negotiation: A comparison of Brazil and the US. *International Journal of Intercultural Relations*, 22, 67–83.
- Peleg, G., Katzir, G., Peleg, O., Kamara, M., Brodsky, L., Hel-Or, H., et al. (2006). Hereditary family signature of facial expression. *Proceedings from the National Academy of Sciences*, 103(43), 15921–15926.
- Russell, J. A. (1994). Is there universal recognition of emotion from facial expression? A review of cross-cultural studies. *Psychological Bulletin*, 115, 102–141.
- Russell, J. A., Suzuki, N., & Ishida, N. (1993). Canadian, Greek, and Japanese freely produced emotion labels for facial expressions. *Motivation & Emotion*, 17(4), 337–351.
- Schwartz, S. H. (2004). Mapping and interpreting cultural differences around the world. In H. Vinken, J. Soeters, & P. Ester (Eds.), *Comparing cultures, dimensions of culture in a comparative perspective* (pp. 43–73). Leiden, The Netherlands: Brill.

- Sorce, J. F., Emde, J. J., Campos, J. J., & Klinnert, M. D. (1985). Maternal emotional signaling: Its effect on the visual cliff behavior of 1-year-olds. *Developmental Psychology*, 21, 195–200.
- Tajfel, H. (1982). Social psychology of intergroup relations. *Annual Review of Psychology*, 33, 1–39.
- Tcherkassof, A., & de Suremain, F. (2005). Burkina Faso and France: A cross-cultural study of the judgment of action readiness in facial expressions of emotion. *Psychologia: An International Journal of Psychology in the Orient*, 48(4), 317–334.
- Triandis H. C. (1995). *New directions in social psychology: Individualism and collectivism*. Boulder, CO: Westview Press.
- Triandis, H. C., Bontempo, R., Villareal, M. J., Asai, M., & Lucca, N. (1988). Individualism and collectivism: Cross-cultural perspectives on self-ingroup relationships. *Journal of Personality & Social Psychology*, 54(2), 323–338.
- Tsai, J. L., & Levenson, R. W. (1997). Cultural influences of emotional responding: Chinese American and European American dating couples during interpersonal conflict. *Journal of Cross-Cultural Psychology*, 28, 600–625.
- Wagner, H. L., & Smith, J. (1991). Facial expression in the presence of friends and strangers. *Journal of Nonverbal Behavior*, 15, 201–214.
- Wheeler, L., Reis, H. T., & Bond, M. H. (1989). Collectivism-individualism in everyday social life: The middle kingdom and the melting pot. *Journal of Personality and Social Psychology*, 57, 79–86.
- Yamaguchi, S. (1994). Collectivism among the Japanese: A perspective from the self. In U. Kim, H. C. Triandis, C. Kagitcibasi, S. C. Choi, & G. Yoon (Eds.), *Individualism and collectivism: Theory, method and applications* (pp. 175–188). Thousand Oaks, CA: Sage.

Chapter 9

Vocal Expressions of Anger

James A. Green, Pamela G. Whitney, and Gwen E. Gustafson

Abstract The empirical literature demonstrates that vocal expressions of Anger are recognized at levels far exceeding chance. Further, several acoustic features of Anger expressions, including intensity, rate, and pitch, appear to serve as the basis for recognition. The standard experimental paradigm for demonstrating recognition of Anger involves actors creating the vocal expressions, pre-selection of stimuli and judges, and forced-choice paradigms for the judges to respond. The possible limitations of this kind of approach are reviewed along with the dominant theories of emotions that are behind the empirical studies. Recent work on embodied cognition as it relates to emotional expressions and suggestions for future studies is discussed.

9.1 Vocal Expressions of Anger

“Go to the window, open it, stick your head out, and yell, ‘I’m mad as hell, and I’m not going to take it anymore.’” Howard Beale, the newscaster in the movie *Network*, is angry, and he believes his viewers ought to be, too, so he exhorts them to yell about it. Yelling, shouting, and screaming are readily recognized as angry speech – in fact, shouting is considered one of the least objectionable forms of aggression across different cultures (Ramirez, 2001, Schieman, 2010). In addition to expressing anger, the voice is also recommended as a tool to *calm* angry feelings. When faced with a potentially Anger-provoking situation, we should, as recommended by the American Psychological Association, “slowly repeat a calm word or phrase such as ‘relax,’ ‘take it easy.’ Repeat it to yourself while breathing deeply” (APA, 2007). Note the emphasis not only on the content of the speech (i.e., use a word like “relax”) but on speed (repeat *slowly*) and at least implicitly on volume (calmly repeat the word).

The linkage between shouting and Anger has, of course, a basis in the empirical literature. Speech identified as angry has generally been found to be fast, loud, and often has a rising melody type. Further, Anger can be identified at above chance levels from vocal expressions alone, and there is cross-cultural generality in the ability to recognize Anger from speech alone. These general findings have led many researchers in the field to the conclusion that specific emotions (e.g., anger, fear, joy) are the result of specific physiological conditions, conditions which give rise to specific patterns of energy in the acoustic waveform of the vocalizations produced during those emotions. One of the

J.A. Green (✉)

Department of Psychology, U-1020, University of Connecticut, Storrs, CT 06269, USA

e-mail: james.green@uconn.edu

pioneers in this field, Klaus Scherer, argued recently that “Given the high recognition of emotions in speech, there must exist emotion-specific acoustic patterns” (Scherer, 2000, p. 227).

In this chapter, we will examine critically the findings on which these general conclusions are based. As one might expect, this tidy state of affairs belies some complex issues about the methods used to generate these data, the interpretation of the findings, and the theoretical approaches guiding the research. The present chapter will take a somewhat different tack from other recent reviews of vocal expressions of emotion (see, e.g., Bachorowski & Owren, 2003; Johnstone & Scherer, 2000; Murray & Arnott, 1993; Russell, Bachorowski & Fernández-Dols, 2003; Scherer, Johnstone, & Klasmeyer, 2003). Of course, we will focus on Anger. In the first section, we will summarize some of the major empirical studies, including cross-cultural studies, but we will then reanalyze some of the published data in order to examine more systematically the errors people make in judging vocal expressions of emotion. We will next explore the general theoretical framework underlying the empirical studies. Although there are vigorous theoretical controversies within the emotion expression literature, there are also some emerging trends in the literatures on general cognitive and motor processes which may be important to future empirical studies on vocal expressions of Anger and other emotions. We review these trends in the last section of the chapter.

For purposes of the initial review of the literature, we will adopt generic definitions of both “anger” and “emotion,” saving more detailed discussion until the latter sections. Further, we will only provide enough information about acoustic analyses of vocal expressions to explain the findings we review. There are several good introductions to acoustic analyses to which interested readers may turn (e.g., Denes & Pinson, 1973; Kent & Read, 2002; Owren & Bachorowski, 2007).

9.2 Empirical Findings on Vocal Expression of Anger

Most of the empirical literature on vocal expressions of Anger follows a similar paradigm: professional actors speak material under instructions to convey different emotions. The spoken material is prescreened for quality before it is played to judges, who select what emotion is being conveyed from a set of emotion terms. In some studies, acoustic analyses of the material are undertaken in order to relate acoustic properties to conveyed emotion. As noted in prior reviews of this literature (e.g., Bachorowski & Owren, 2003), decisions at every step in this methodology have the potential to affect (some would say “bias”) the findings. We shall examine some of these methodological issues in detail after reviewing a few of the major empirical studies. (For the convenience of the reader, Table 9.1 contains a brief listing of the studies to be reviewed and some of their important methods and results. A much more extensive table can be found in Juslin and Laukka (2003).)

9.2.1 *Selected Studies*

The earliest experimental study of emotional expressions and vocal qualities is likely the 1936 paper by Fairbanks and Provonost. More than 70 years ago, this study used “six competent actors” to portray five different emotions – Contempt, Anger, Fear, Grief, and Indifference. A common passage of text, three sentences long, was embedded in different stories, which were read by the actors and recorded on phonograph records. The records were played to a class of advanced speech students, who chose one of the 12 emotions as characteristic of the passage. The Anger passages (one each from the six actors) were judged to be Anger by 78% of the speech students. (No statistics were present in this classic paper, but clearly this is far above the 8.3% chance level of responding correctly.) When Anger was confused with one of the other 11 emotions, it was most often judged to be Contempt or Jealousy.

Table 9.1 Summary of empirical studies reviewed

Study	Coders	Decoders	Task type	Number of emotions	Outcomes
Banse & Scherer (1996)	12 actors	12 students	Forced choice, 12 alternatives	14	Hot anger recognized the best at 78%. Emotion category was able to predict most acoustic parameters using multiple regression
Fairbanks & Pronovost (1936)	6 acting students	64 speech students	Forced choice, 12 alternatives	5	Anger recognized above chance. Anger expressions higher pitched
Juslin & Laukka (2001)	8 actors	15 students	Forced choice 5 alternatives and rating of how similar the emotions are to each other	5	All emotions recognized above chance. Anger most often mistaken for disgust. But Anger not rated as similar to any other emotion
Scherer et al. (2001)	4 actors	428 subjects from 9 different countries	Forced choice 5 alternatives Nonsense words used a stimuli	5	Anger was recognized the best. Ratings and errors similar for respondents from all countries
Sobin & Alpert (1999)	31 female	12 female	Rate the presence of a given emotion on a 7-pt scale	4	Anger most often mistaken for fear. Anger expressions were fast, high in intensity, and low in F_0
Van Bezooijen et al. (1983)	8 students	48 Dutch 40 Taiwanese 41 Japanese	Forced choice 10 alternatives, Sentences presented In Dutch	10	All emotions Recognized above chance. Taiwanese and Japanese participants showed significantly lower recognition rates. Activity appears the strongest dimension for cross-cultural recognition of emotions

Pitch determination of the emotion portrayals was the focus of the acoustic analyses in this study. (Pitch was apparently measured from the waveforms of the speech on the phonograph records and is typically assumed to be the fundamental frequency or F_0 of the sound.¹) The median pitch of Anger was 228.8 Hz, lower than that for Fear (254.4 Hz), but higher than Contempt, Grief, and Indifference (124.3, 135.9, and 108.3 HZ, respectively). Fairbanks and Provonost also reported large

¹ The *fundamental frequency* of a speech sound corresponds to the rate at which the vocal cords are opening and closing. For adults, F_0 is typically in the 100–200 Hz (or cycles per second) range. Middle C on the piano has a pitch of 220 Hz; our ability to distinguish a voice from a piano note lies large in the other frequencies, or harmonics, that are generated by the vibration source and which then are emphasized by the resonators set in motion by the vibrating source. *Pitch*, on the other hand, is a perceptual characteristic; perceived pitch is usually based on the fundamental frequency of the voice but there are conditions under which this relation does not hold.

pitch changes, and a wide range of pitch, during portrayals of Anger. The authors concluded that emotions expressed by voice were identifiable and that pitch characteristics of the speech signal distinguished the emotional portrayals.

Two findings from this 1936 study have been particularly robust. One is that, when a forced-choice response paradigm is used, vocal expressions of Anger are judged as Anger at above chance levels. (A forced-choice paradigm is one in which judges must select an emotion term from a pre-defined list of terms.) The second finding is that average pitch was higher in Anger expressions than in expressions of Grief or Indifference. These findings have subsequently been confirmed in several studies (Banse & Scherer, 1996; Leinonen, Hiltunen, Linnankoski, & Laakso, 1997; see also the review by Murray & Arnott, 1993, of earlier studies).

Sixty years after the Fairbanks and Provonost study, Banse and Scherer (1996) published a major and oft-cited study of vocal expressions of emotion and their relation to acoustic attributes of speech. To obtain the vocal stimuli for this study, 12 actors were asked to speak two nonsense phrases, twice, for each of 14 emotions in each of two scenarios. The 14 emotions were selected to cover a wide range and included pairs similar in valence (positive versus negative) but differing in intensity (e.g., happiness versus elation, sadness versus despair). Of the 1,344 total voice samples, the 280 best recognized portrayals of each emotion were selected by 12 additional trained actors. These stimuli were then played to 12 undergraduate psychology students, who chose one emotion out of 14 in a forced-choice procedure. The stimuli were analyzed acoustically and nearly 30 acoustic parameters were measured, including fundamental frequency (F_0), energy distribution, and speech rate.

Recognition rates for the 14 emotions ranged from 15 to 78%, with a mean percent correct of 48. (Compared with chance performance, 12 of 14 were significant.) "Hot Anger" was recognized correctly 78% of the time and "Cold Anger" 34% of the time. The authors used multiple regression to predict each of the acoustic variables from sets of dummy-coded variables, including one set for the 14 emotion categories. Emotion category significantly predicted 25 of 29 acoustic parameters. Predictions for which acoustic features would characterize each emotion were made based on earlier theory (Scherer, 1986) and were confirmed about half of the time. The authors interpret their findings as support for the ideas that humans can infer specific emotions from vocal expression alone and that Scherer's component process theory (reviewed below) makes correct predictions about the acoustic features of emotional expressions encoding the specific emotions.

Sobin and Alpert (1999) attempted to rectify several methodological shortcomings of previous studies, including Banse and Scherer (1996), shortcomings which they believed had led to the "decoders" of vocal expressions of emotions having greater accuracy than could be achieved only on the basis of the acoustic attributes of the expressions. These shortcomings included small samples of vocal expressions, heterogeneity of the emotion "encoders," the use of nonsense speech to express emotions, and the use of forced-choice rating systems to collect data. Sobin and Alpert used 31 emotion "encoders" in this study and 12 emotion "decoders." All were female. A single sentence was embedded in 20 different, 300-word stories, with five stories for each of the four emotions of Fear, Anger, Joy, and Sadness. This embedded sentence paradigm was believed to yield stronger induction of emotions in the encoders and therefore more veridical acoustic characterization of emotions. Four sets of three decoding "teams" were trained by the authors as to definitions of their assigned emotion and "the ways that it might affect speech." Each rater made ratings of a single emotion for each of 375 sentences (15 different encoders producing the 20 different sentences and an additional five neutral sentences), using a 7-point scale of expressed intensity. Twelve acoustic variables were measured for each sentence, including average intensity and pitch measures as well as several measures related to speech rate and pauses. The acoustic measures were taken on 152 sentences selected as "prototypical," that is, as scoring high on the intended emotion and low on all of other emotions.

Sobin and Alpert found that sentences spoken by encoders as Fear and Sadness were rated as more intense than those spoken as Joy and Anger. As a measure of emotion recognition, Sobin and Alpert calculated the percent of tokens which were given their highest intensity rating on an emotion other than what had been intended. Only 17% of total number of sentences spoken by encoders fell into this category. (When it was misperceived, Anger was most often misperceived as Fear, 14 out of 155 tokens, but sometimes as Joy, 6 out of 155 tokens.) Eight of the 12 acoustic attributes differed among the four emotions, with Anger being characterized as fast, high intensity speech, but *low* in F_0 (the opposite finding from Fairbanks and Pronovost, 1939 and Banse and Scherer, 1996). Multiple regression indicated that 45% of the variance in Anger ratings was accounted for by the 12 acoustic attributes. The authors attributed the failure to replicate past work on F_0 and Anger to the use of all female decoders and perhaps to the use of nonactors as encoders. They suggest future studies enlarge the range of acoustic variables and include other sets of decoders (e.g., males).

Two final examples from this literature examine cross-cultural similarity in emotion recognition. Scherer, Banse, and Wallbott (2001) used four professional actors (two female and two male) to express five basic emotions, Joy, Anger, Fear, Anger, and Disgust. The embedded sentence method was employed, in that a standard sentence was embedded in a vignette that served to evoke different emotions. Because the study was cross-cultural and cross-linguistic, nonsense sentences were constructed out of the syllables of six European languages. The two sentences judged to be most "sentence-like" were embedded in the vignettes. (One of these sentences, for example, was "Hat sundig pron you venzy.") Thus, the study design was factorial, with four actors, two scenarios, five emotions, and two sentences generating a total of 80 utterances. Each sentence was also uttered in a neutral manner.

A final stimulus set was chosen by Scherer et al. that included 30 utterances judged by university students as good exemplars of one of the five expressed emotions. In the end, Disgust was dropped from the final set because it was too often confused with Anger and Sadness. There were slightly unequal numbers of sentences representing the emotions, the judges, and the two sentence varieties. University students in nine different countries judged the emotion spoken in a forced-choice paradigm.

Judges from all countries made choices that agreed with the intended expression more often than chance; kappa values ranged from .37 (Indonesia) to .69 (Germany). Working with an accuracy score, analysis of variance showed main effects of both emotion and country. The only significant mean difference between countries was in comparing Germany and Indonesia. However, additional analyses showed significant interactions between country, gender of the actor, and emotion, indicating that there were differences across countries in how specific emotions spoken by specific actors were judged. These effects were generally smaller than the overall country effect, however, and were not discussed by the authors. Anger was the best recognized of the emotions, with 77% accuracy. Joy was the most poorly recognized, with 42% accuracy.

In their Discussion, Scherer et al. (2001) raise the possibility that the country differences in their study were grounded in linguistic differences. The countries with languages less similar to German performed more poorly. Although the authors argue that the use of nonsense sentences must have forced attention to nonlinguistic cues, these interactions involving country/linguistic effects could be viewed as damaging to the hypothesis of a generic physiological substrate of Anger and its effects on the nonlinguistic aspects of speech.

A different cross-national study (van Bezooijen, Otto, & Heenan, 1983) also found that nonnative speakers performed more poorly at recognizing vocal expressions of emotions. Eighty tokens of the Dutch phrase "two months pregnant" were portrayed vocally in 10 different emotions by eight students at the University of Nijmegen. Student judges from Holland, Taiwan, and Japan

listened to the tokens (all in Dutch) and selected the appropriate emotion using the standard forced-choice paradigm. Correct recognition was significantly poorer in Taiwan and Japan than in Holland, although all the emotions were recognized above chance levels. Multidimensional scaling was used to attempt to uncover the dimensions underlying confusions of each emotion with every other emotion. The authors concluded that there was clearly an “activity” dimension underlying the judgments, as well as a second dimension that could not be interpreted.

9.2.2 Summary and Limitations

The studies reviewed reveal several consistencies in findings: First, when the Anger expressions are spoken and decoded by adults who share the same language, recognition of Anger in a forced-choice paradigm is far above chance – 78% in Fairbanks and Provonost (1936), 78% in Banse and Scherer (1996), 76% in Sobin and Alpert (1999), 79% in Scherer et al. (2001), and 67% in van Bezooijen et al. (1983). The comparability of these figures would be nearly impossible to duplicate in other areas of behavioral science; these figures are most impressive. Second, there is some consistency in the emotions that are most often confused with Anger; Contempt is the most common incorrect choice in three of these studies (Banse & Scherer, 1996; Fairbanks & Provonost, 1936; and van Bezooijen et al., 1983), but Joy is a common incorrect choice as well (Scherer et al. in the Indonesian sample and van Bezooijen et al. in the Taiwanese and Japanese samples). Of course, one often hears the expression “shouting for joy,” but the fact that Joy expressions are one of the most often confused with Anger expressions deals a rather serious blow to the notion that vocal expressions are closely linked to the underlying, experienced emotion of speakers. Finally, cross-cultural studies consistently show better recognition when speakers and decoders come from the same country (Scherer et al., 2001; van Bezooijen et al., 1983), and this same intracultural advantage has been found in the identification of facial expressions (Chapter 8 by Matsumoto et al., this book.)

However, all of these studies have serious limitations, many of which have been raised by the authors themselves. A brief list includes (1) the use of professional actors to generate emotion tokens, (2) the pre-selection of the best tokens for subsequent analysis, (3) the use of the forced-choice methodology by the judges, (4) the diversity of tokens selected for presentation, (5) the methods used to elicit Anger and other emotions, and (6) the influence of language and cultural norms and practices on the production and perception of vocal expressions. These limitations have been discussed in detail in recent reviews of the literature (e.g., Bachorowski & Owren, 2003; Johnstone & Scherer, 2000; Murray & Arnott, 1993; Russell et al., 2003), and they are, to some extent, a predictable consequence of the general theoretical perspective underlying the research. We will return to some of these issues below, but now we follow up one in particular, namely, the use of forced-choice methodology.

9.3 Confusions Among Anger and Other Emotion Terms

Many of the studies reviewed above present confusion matrices as part of their results sections. Thus, it is possible to examine the percent of time that Anger was judged as Anger but also the percent of time that Anger was judged as Disgust, Joy, and so on. Despite the availability of these confusion matrices, however, only van Bezooijen et al. (1983) used a quantitative technique to analyze the structure in judges’ perceptions of vocal expressions of Anger or other emotions. Below, we illustrate how to use multidimensional scaling procedures to examine the relation between Anger and other

judgments in these studies as well as to gain some insight into the contribution of the forced-choice method to the structure in judges' responses.

Consider the data in Table 9.2, adapted from Juslin and Laukka (2001). This table shows the number of times participants judged portrayals of Anger as Anger ($n = 58$), portrayals of Anger as Disgust ($n = 31$), and so on. Recall that the data came from a forced-choice paradigm. Although simple inspection of this matrix is informative (especially because it is relatively small!), using the matrix as input to multidimensional scaling (MDS) generates a more comprehensible visual representation of the closeness of these emotion portrayals.

Table 9.2 Emotion judgments using forced-choice method (adapted from Juslin & Laukka (2001), with permission of the author)

Emotion portrayed	Emotion judged					
	Anger	Disgust	Fear	Happiness	Sadness	None
Anger	58					
Disgust	31	40				
Fear	0	2	60			
Happiness	6	3	21	51		
Sadness	1	2	24	1	63	
None	4	4	4	5	11	72

Note. The "None" category represents actors portraying no emotion during their speech tokens (see Juslin & Laukka, 2001).

Figure 9.1a shows the two-dimensional solution from a standard MDS program (see Kruskal & Wish, 1978, for a general Introduction to MDS). Note how close Anger is to Disgust in the configuration; this makes perfect sense as the largest number of confusions was between Anger and Disgust (Table 9.1). In MDS, the distance between objects in the two-dimensional space corresponds to the number of confusions in the data matrix. The closeness of Disgust to Anger could be interpreted as a blow to theories that posit a small number of discrete, primary emotions. Recall, however, that judges were forced to choose one of the alternatives, so the closeness of Disgust and Anger could be the result of Disgust being more similar to Anger than to either Fear, Happiness, or Sadness. When judges *must* choose one emotion from a limited set, and when they choose the inappropriate emotion, we have been provided with information about only two emotion terms, the target and the most similar term from the set.

In a wonderful addition to the standard paradigm, Juslin and Laukka also asked participants to *rate* each portrayal on the attributes of Anger, Disgust, Fear, Happiness, Sadness, or No Emotion Expressed. Thus, judges gave information about the closeness of each emotion portrayal to *every other* emotion term in the set, not just to one other as in the forced-choice method. Figure 9.1b shows a two-dimensional MDS solution for these rating data. (Note that the similarity among rated attributes is calculated as either the Euclidean distance or the correlation between them.) Note that Fear and Sadness are now judged the most similar emotions (using rating data), whereas Anger is relatively isolated and Disgust has moved closest to the No Emotion judgment.

Thus, the forced choice and the rating data (Fig. 9.1a, b) yield somewhat different configurations of the emotions in the "perceptual space" of the judges. It appears that concerns raised in previous reviews about the almost exclusive reliance on forced-choice paradigms are indeed valid ones. We would argue that one of the primary limitations of the forced-choice paradigm is that judges' errors only reflect a judgment about one *pair* of the emotions in the set. Ratings of each emotion portrayal's similarity to every other emotion yields a different, arguably richer picture of judge's perceptions. In these data, the general topography of the mappings is similar (Fig. 9.1a, b), but the relative distances

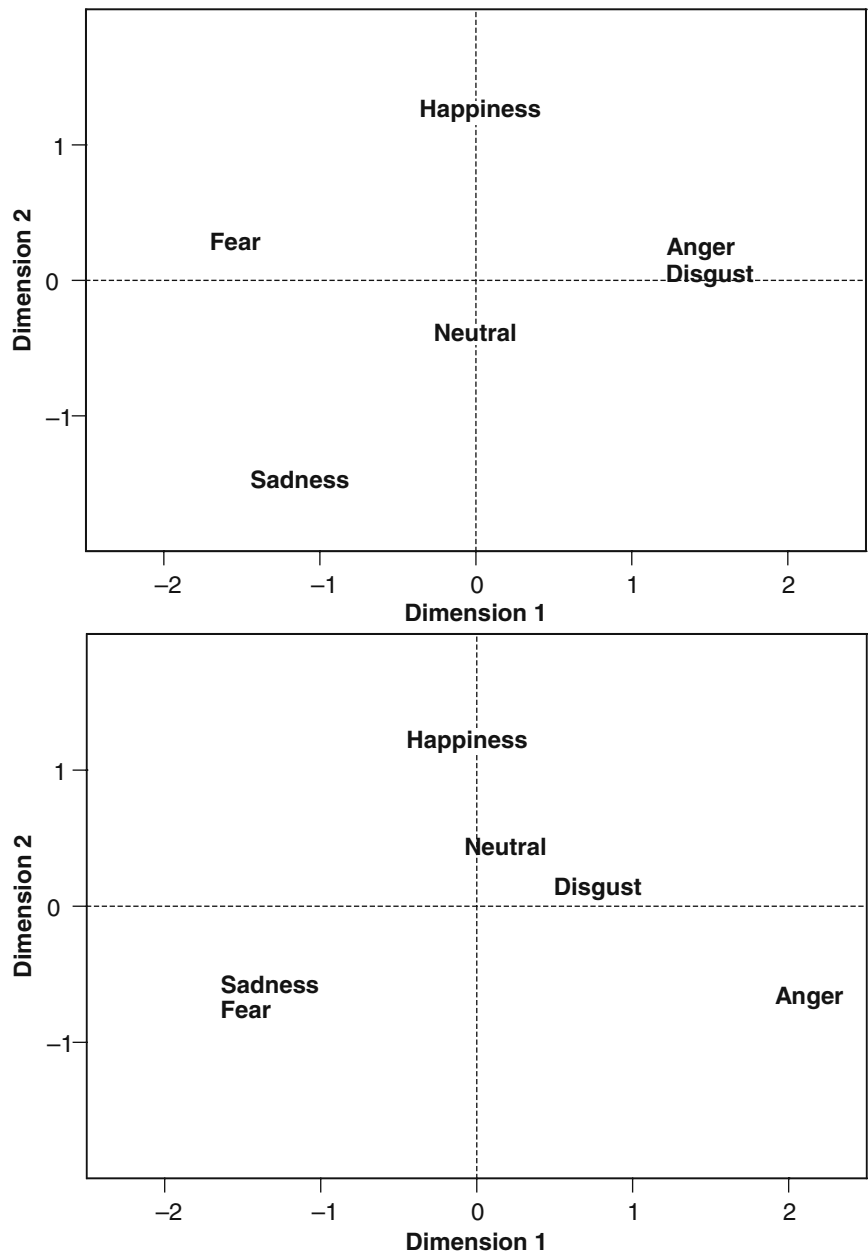


Fig. 9.1 Panel A (the *top* figure) is the spatial representation of confusions in Table 9.1 (with arbitrary dimensions and units). Panel B (the *bottom* figure) is the spatial representation of the same emotions based on ratings data (with arbitrary dimensions and units)

between pairs of emotion terms differ. In this case, Anger is relatively distant from the other five emotion portrayals when ratings are employed, although the closest emotion to Anger using both methods is Disgust.

Confusion matrices like those in Table 9.2 are small, and it is relatively easy to discern patterns in the data; however, systematic methods of finding patterns are necessary when the number of stimuli becomes larger. Consider the data in Table 9.3, from Banse and Scherer (1996). Reading down the first column, we see that Hot Anger (HA) was indeed most often judged to be Hot Anger by the participants in the forced-choice paradigm (78% of the time). However, it was confused with Cold Anger 10% of the time and with Contempt 11% of the time. Panic portrayals were judged to be Panic 36% of the time, but to be Anxiety 27%, and Despair 21%, of the time.

Table 9.3 Emotion judgments using forced-choice method (adapted from Banse & Scherer (1996, with permission of the author)

Emotion judged	Emotion portrayed													
	HA	CA	PC	AX	DE	SD	EL	HA	IN	BO	SH	PR	DI	CN
HA	78	17	10	0	6	0	14	0	0	0	0	0	0	2
CA	10	34	2	7	5	1	5	1	3	2	2	5	10	10
PC	0	0	36	13	9	1	7	0	0	0	1	0	1	0
AX	0	0	27	42	18	2	5	1	1	0	15	2	10	2
DE	0	0	21	7	47	21	16	1	0	1	4	0	10	0
SD	0	0	0	5	8	52	0	3	0	13	19	2	14	8
EL	0	1	0	0	1	0	38	2	0	0	0	4	0	0
HA	0	2	0	4	1	3	1	52	8	1	8	23	5	0
IN	0	7	1	7	0	1	4	18	75	1	13	12	2	2
BO	0	4	0	1	0	5	0	4	1	76	4	2	2	4
SH	0	0	1	5	2	9	1	3	1	1	22	2	10	2
PR	1	15	1	4	0	2	2	17	10	1	8	43	7	6
DI	1	2	1	2	2	0	2	0	1	0	1	0	15	5
CN	11	18	1	4	3	3	7	1	1	6	4	6	15	60

Banse and Scherer suggested that there are three dimensions evident in these confusions: *quality* (hot versus cold anger, elation versus happiness, sadness versus despair), *intensity* (e.g., despair, hot anger, and panic are all high intensity), and *valence* (the usual positive versus negative emotions). However, this description of the dimensions underlying judges' perceptions was qualitative only. Fortunately, because the confusion matrix was published, we can use quantitative techniques, as in the Juslin and Laukka (2001) analyses above, to look for patterns in the confusions. MDS was used to explore the dimensionality of a 14×14 matrix representing the similarity of all pairs of the 14 emotions. (The proximity was calculated by computing the distance between each pair of columns in Table 9.3 across the 12 emotions not in the pair. That is, the similarity of Hot Anger and Cold Anger was considered to be how similar their profiles of confusions were across Panic, Anxiety, Despair, and so on.)

Applying MDS to the matrix of similarities showed that the best fitting configuration was, indeed, three dimensional (stress = 0.08, see Kruskal & Wish, 1978). However, Banse and Scherer's analysis suggesting attributes of quality, intensity, and valence does not fully map onto this configuration (Fig. 9.2). The same emotions but with different qualities are not always close together in this space. For example, although Hot Anger and Cold Anger occupy a similar space, Happiness and Elation do not, nor do Panic and Anxiety. The attribute of intensity does not appear to be represented in this space either. Panic and Hot Anger, both very intense emotions, are very far apart in the space and do not lie along any single direction through this space.

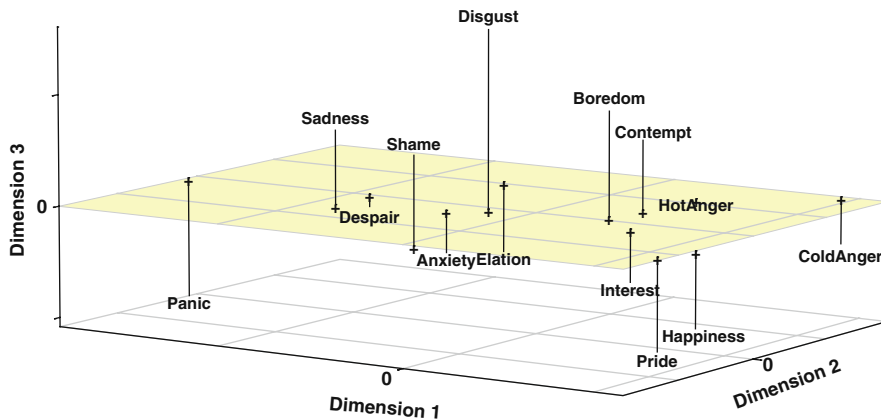


Fig. 9.2 Spatial representation of the similarity of emotion portrayals in Table 9.2

The simple valence of these emotion terms is the one attribute Banse and Scherer mention that might map onto the configuration; the positive terms of Interest, Happiness, and Pride are relatively low on Dimensions 2 and 3 but high on Dimension 1. Most of the negative emotions have higher scores on Dimensions 2 and 3 and lower scores on Dimension 1. To test this notion quantitatively, the emotion terms were assigned a +1 or -1 depending on their positive or negative valence. Then, this dummy coded variable was regressed onto the three dimensions of the configuration (see Kruskal & Wish, 1978, for a discussion of this vector fitting analysis). Valence appeared to fit into the configuration reasonably well: the final R^2 value was, .73, and the beta weights suggested that a valence vector was most closely aligned with Dimension 3. In the next section, we will review *core affect theory*, which argues that valence and arousal are the two basic dimensions underlying expressions of emotion. Obviously, the MDS analysis shown here is compatible with a dimensional theory, and the valence of these 14 emotions appears to one of the attributes that is salient to participants as they select alternatives in the forced-choice paradigm.

From these secondary analyses of the Juslin and Laukka (2001) and Banse and Scherer (1996) data, we can make two major points. First, the forced-choice paradigm gives a different view of the perceptual space of Anger, and other emotions, than does a rating task. Anger is quite often confused with Disgust in a forced-choice task (Fig. 9.1a) but is rated quite differently from Disgust in a Likert-type rating task (and Disgust is rated similarly to a Neutral emotion). We argue that these differences are because the forced-choice method gives less complete information about the emotions being portrayed than other methods (e.g., rating each portrayal on each of several emotions). The second major point to be made is that quantitative techniques are available to evaluate confusions and similarities among emotion portrayals. We employed multidimensional scaling, but cluster analysis is also appropriate; indeed, these techniques are often employed together to yield a more complete picture of the objects in question (see Kruskal & Wish, 1976). The use of these approaches as secondary analysis techniques raises the question of what other types of data gathering procedures might be employed? MDS and clustering are particularly useful for *paired comparison* data, and it might be useful to begin to employ this type of data collection in the study of vocal expressions of Anger and other emotions. Then, the similarity of Anger to each portrayed emotion can be directly judged and individual or group perceptual spaces can be generated and compared directly.

9.4 Theories Related to the Vocal Expressions of Emotions

9.4.1 The Classic View

The methods used to study vocal expressions of Anger are intimately linked to the theory guiding the research, and it is therefore useful to review several of the theoretical approaches to vocal expressions of emotion. For example, recall that there is less-than-perfect matching in the empirical studies reviewed above, and a heavy reliance on forced-choice methods. In order to understand why this pattern recurs over studies, it is useful to look at the dominant theories in the field. By doing so, we hope to open up new possibilities for research on vocal expressions of Anger and perhaps encourage a different view of the current set of findings.

9.4.1.1 Discrete Emotions

Discrete emotions theory posits a finite set of distinguishable, coherent physiological states that map, one-to-one, onto different expressions. As Scherer et al. (2003) note, these mappings have been worked out most thoroughly for facial expressions and scant attention has been paid to predictions about vocal expressions of emotions. However, it would seem that the emotions as experienced, if they are linked to discrete physiological states, ought to have predictable consequences for vocalizations. Anger in response to threat, for example, should involve sympathetic arousal, increased heart rate, and increased muscle tone (Scherer et al., 1986). These changes could help explain the increased tempo of angry speech, the increased loudness, and the increase in F_0 . Along these lines, Siegman has emphasized linkages between the experience of Anger and physiological conditions predisposing to cardiovascular disease (e.g., Siegman & Snow, 1998).

9.4.1.2 Component Processes

The best articulated “classic” theory of vocal emotion expression has been provided by Klaus Scherer, termed the *component process theory* of emotions (see reviews by Scherer, 1986, 2003; Scherer et al., 2003; Wranik & Scherer, 2010). According to this theory, organisms are constantly scanning their external and internal environments and performing “stimulus evaluation checks” or SECs. These SECs include checks for novelty, intrinsic pleasantness, goal/need significance, coping potential, and norm/self compatibility. Each outcome of an SEC will have a specific effect on the somatic nervous system (SNS). As Scherer (1986) puts it, “the characteristics of the vocal expression at a particular time are the *net result* of the effects of the outcomes of the preceding SECs in the information processing subsystem and of the total effect of the changes in the other subsystems impinging on the SNS” (p. 148, original italics).

For Scherer, one of the reasons for the loose connection between acoustic cues and vocal expressions of emotion is the failure of studies to take into account the differential cue structure associated with different configurations of SECs that have been performed. (Scherer also argues that the correct acoustic features have not always been measured.) He distinguishes, for example, between cold and hot anger, with cold anger associated with low novelty and only medium relevance to goals and urgency, whereas hot anger is the result of high novelty and high relevance and urgency. Other emotions are often split into two based on urgency or novelty (e.g., joy is subset into happiness versus elation and displeasure into contempt and scorn). Note, however, that this theoretical distinction did not help explain the confusions in the Banse and Scherer (1996) data shown in Table 9.3 and Fig. 9.2. Hot and Cold Anger are often confused, but Happiness and Elation are rarely confused by judges.

Scherer et al. (2003) take pains to distinguish this theory, which they call an *appraisal theory*, from the discrete emotions theory reviewed above. The major distinction, at least for the emotion of Anger, would appear to be what drives the unique physiological configuration underlying the angry expression. Scherer et al. suggest that discrete emotions theory posits “innate neuromotor” programs as the underlying mechanisms, whereas it is the appraisal of the environment that leads to the underlying physiology in the component process theory. Both theories, however, would argue that the vocal expression of Anger depends almost completely upon the configuration of the underlying physiological state.

9.4.2 Alternatives to the Classic View

9.4.2.1 The Core Affect Approach

Quite different from the discrete emotions or the component process view, the core affect approach suggests that an “*emotion* refers to a heterogeneous cluster of loosely related events, patterns, and dispositions” (Russell, 2003, p. 167). Russell argues that the “primitives” in most theories of emotions are signified by words like anger, sadness, joy, and so on. These are the fundamental, experienced, and appraised feeling states or basic units of most emotion theories.

Russell proposes, however, that the first primitive in a theory of emotions ought to be the combination of two underlying *dimensions* of *valence* and *arousal* which Russell terms *core affect*. Humans always have core affect in a “floating baseline” which is accessible to consciousness but not cognitive in character. “Core affect is that neurophysiological state consciously accessible as the simplest raw (nonreflective) feelings evident in moods and emotions” (Russell, 2003, p. 148).

The second primitive in the core affect theory is the perception of affective quality or the perception of the valence and activation of the core affect. We do not have unfettered access to core affect; sometimes our attributions about changes in our core affect are simple (e.g., being afraid of a dog charging, or being angry at a boss) and sometimes we have limited access to what caused a change in our core affect. Clearly, Anger would occupy a position in the core affect system that would be high in arousal and negative in valence.

In Russell’s (2003) own words, what he hopes to achieve is “a synthesis of (a) James’s (1884) insight that emotion involves a self-perception of automatic processes with (b) modern evidence on the processes involved” (p. 146). Changes in core affect (i.e., in arousal and valence) represent changes in neurophysiological states. We have some conscious access to these changes but the access is not perfect.

Russell’s core affect approach is very similar in character to Buck’s approach to “biological affects” (Buck, 1999). Buck argues that the discrete, differential emotions and appraisal theories are top-down theories, with emotions being the *result* of cognitive appraisal of environmental events. Buck argues instead that emotions are “readouts” of phylogenetically structured, special-purpose neurochemical systems. Like Russell, Buck argues that these systems are always active, and that humans can, if they choose to, attend to them at any moment in time. When these systems become active due to encounters with challenging stimuli, our autonomic, endocrine, and immune systems respond in efforts to adapt. Further, we have subjective experiences of these changes, and we have expressive displays, centered on the face, that are innate but which can be modifiable by cultural and situational display rules. Anger, for Buck, is a biological affect associated with the limbic system, and it is fundamentally agonistic in character because of its association with situations of competition, conflict, and fighting (see also Whalen, Shin, & McInerney, 2001). (However, recent neuroimaging studies suggest an important role for cortical structures, especially the orbitofrontal

cortex and anterior cingulate gyrus, in response to facial expressions of anger and to transient induction of anger, see Blair, Morris, Frith, Perrett, and Dolan, 1999, and Kimbrell et al., 2001.)

9.4.2.2 The Functionalist Approach

Rather than defining emotions as either discrete states or as underlying dimensions, the functionalists discuss “emotion families.” The families have “fuzzy boundaries” and are based on three adaptive functions: behavior-regulatory, social-regulatory, and internal-regulatory. The behavior-regulatory functions may not be carried out but are always associated with a tendency to act in a certain way. Social-regulatory functions involve social communication and internal-regulatory functions may involve focusing one’s attention on an impending threat. The adaptive functions are rarely observable and, therefore, must be inferred from behavior or reports.

An emotion occurs when an individual appraises a relationship between the self and the environment as being significant. The emotion is viewed as a process unfolding in time, not as a program in the brain; however, the appreciation of a particular relationship may not be conscious and may even be “prewired.”

9.4.2.3 Summary

Russell et al. (2003) provide an excellent review of theoretical issues in the expressions of emotions (EEs). Arguing, as we have above, that emotions have historically been considered discrete states with coherent components – physiological conditions, facial and vocal expressions, subjective experiences, and instrumental actions – Russell et al. suggest that the empirical efforts to find EEs within this framework have met with only limited success. They suggest that there may be limited universality in the decoding of EEs, especially faces, and that the decoding of emotions by receivers is not just a cognitive exercise. Rather, emotions have evolved to influence social partners; thus, EEs may function to garner the attention of conspecifics (and perhaps predators) and to increase their arousal.

In addition to advancing our understanding of what “decoding” an emotion entails, Russell et al. point out that there is very little empirical work on how emotions in the “sender” are actually related to expressions. As we have seen in the empirical studies reviewed above, much of the research involves actors producing EEs. An initial corpus of vocally expressed emotions is usually mined to find the best expressions of anger, joy, sadness, and the like. Clearly, this work relies on culturally shared meaning regarding how an emotion such as Anger is likely to be expressed by voice or face. Russell et al. suggest that the emotional state of the sender in these studies has basically been neglected.

In a strongly worded conclusion, Russell et al. (2003) state “Emotion expressions may not be expressions and may not be related to emotions in any simple way” (p. 342). They argue that new research ought to tackle the question of what processes in decoders are quick and relatively automatic and which require effort and even training. For example, responding to a vocal expression which might signal Anger may involve attention recruitment, an increase in arousal, and the search for situational cues to evaluate both the sender’s condition and the range of possible responses.

Both the core affect and the functionalist accounts predict less clear mappings between experienced Anger, vocal characteristics, and the perception of Anger by listeners. Beyond this notion of a looser coupling of acoustic patterns to underlying emotions and to perceived emotions, more specific predictions are difficult. However, the same can be said of the discrete emotions theory. Only Scherer’s component process theory makes specific predictions about which acoustic features should map onto Anger. As Scherer et al. (2003) have already written, the different emotion theories do not permit highly specific predictions about concomitant vocal expressions, including Anger.

Future empirical studies in this area might benefit from detailing the *perceptual* as well as the acoustic features of anger vocalizations. Schieman (2010) argues that “yelling” is a vocal expression associated with intense feelings of anger; Potegal (2010) has also argued that anger intensity can be captured by perceptual qualities of vocalizations. Scant attention has been paid to how intensity of experienced anger might relate to expression through vocalization, yet this linkage would have important implications for the theoretical positions outlined above.

9.5 Recent Theoretical Advances Relevant to Vocal Expressions of Anger

Russell et al.’s review and the new viewpoints expressed by Buck (1999) and by the functionalists all suggest that conceptualizations of emotions and their expressions are changing, partly as the result of several decades of empirical work and partly as the result of changes in psychological theorizing. We have reviewed some of the empirical work on vocal expressions of Anger and other emotions, but what recent theorizing in general cognitive psychology might be relevant to this emotion literature? This topic is the last one to which we turn.

“Embodied cognition” is one of the new concepts in the general psychological literature that might move the vocal expressions literature in a new direction. Embodied cognition refers, roughly, to an approach to cognitive science that recognizes that human brains are situated in human bodies that perceive and act in the world. This contrasts with the classic view of human brains as symbol manipulating devices. One explicit formulation of this idea was found in Buck’s notion of interoceptive affordances, the idea that we can be aware of changes in our bodily processes through a specialized system of perception. Some of the experimental work done within the framework of embodied cognition might help explain how, and why, Anger is expressed in vocalizations and speech and how that speech is experienced by listeners.

For example, consider the recent report by Niedenthal, Brauer, Halberstadt, and Innes-Ker (2001) on recognition of facial expressions of sadness and happiness. Niedenthal et al. explored the effects of mimicry on recognition of dynamic depictions of facial expressions. In experiment 1, Niedenthal et al. demonstrated an effect of emotional congruence on perception of emotion faces. Movies of faces morphing from happy to sad, or vice versa, were shown to undergraduates whose emotional state was manipulated by a film and music. The participants’ task was to judge the point in the movie when the face no longer expressed the initial emotion (happy or sad). Participants in the induced happiness condition judged the offset of happiness earlier than participants in the induced sadness condition; likewise, participants in the induced sadness condition judged the offset of sadness earlier than participants in the induced happiness condition. In a second experiment, emotion induction was not used; rather, half the participants were asked to hold a pen between their lips and teeth, thus preventing facial mimicry. The frame of the movie in which a change was detected was again used as the dependent variable. The results indicated a main effect of mimicry; participants who could mimic the faces detected a change in facial expression earlier in the movies than participants who could not.

Niedenthal et al. (2001) provide the first evidence that mimicry might be a process important in the recognition of facial expressions of emotion. The authors go on to point out that there are several mechanisms by which mimicry could affect judgments. However, the important point seems to be that both induced emotional state and facial mimicry affect judgments of emotional expressions. Thus, the appraisal, or other cognitive processes involved in making the judgment of an emotional expression, is embedded in a matrix of perception and action. This demonstration is clearly in accord with the notion that cognition is embodied.

How might this idea be applied to vocal expressions of Anger? Clearly, emotional state of the decoders in such studies could be induced, as in the Niedenthal et al. (2001) study, by the kinds of procedures used in previous studies of Anger. It is more difficult to conceptualize a vocal process analogous to facial mimicry that could be employed. Although preventing facial mimicry could affect judgments of vocal expressions of Anger, it would seem more likely that disrupting the processes which might be involved in vocal production would yield more interesting results.

One recent study did examine vocal prosody as an “embodied emotion.” Neumann and Strack (2000) played a 5-min recitation of a text on philosophy to undergraduates, who were to be tested on comprehension of the material. However, the text was read in either a happy, neutral, or sad prosody, and the mood of the participants was evaluated by questionnaire immediately following hearing the passage. Participants who listened to happy prosody reported more cheerful moods than participants in either the neutral or sad condition. In a series of three follow-up experiments, Neumann and Strack attempted to disentangle the effects of listeners’ cognitions about the speaker’s mood from more automatic induction of mood in the listener. The authors suggest that “action codes” are induced in the listeners by emotional prosody and that automatic, nonconscious processes are responsible for at least a portion of this phenomenon (which they refer to as *mood contagion*).

Note that the idea that heard speech results in the activation of “action codes” in listeners is consistent with the *motor theory of speech perception* championed by Liberman (Liberman & Mattingly, 1985) and reviewed recently by Galantucci, Fowler, and Turvey (2006). In this theory, the objects of perception in the process of listening to speech are not the acoustic patterns generated in the air by a speaker but rather the speakers’ vocal tract gestures. The latter are referred to as *phonetic gestures*. In fact, Galantucci et al. go on to review several studies suggesting that perception and action share a common code in the brain/body, and that “the architecture of cognition is permeated by the linkages between the perceptual and the motor systems” (see also Prinz & Hommel, 2002).

How might this be relevant to theories of emotional expression and Anger? First, it appears that expressing Anger and experiencing Anger may share resources in the neural architecture of humans. Thus, the common tendency to neatly separate sender and receiver may be quite artificial. In fact, Carr, Iacoboni, DuBeau, Mazziotta, and Lenzi (2003) used fMRI to show that participants who imitated or watched images of emotional faces had activation in similar brain areas. “We ground our empathic resonance in the experience of our acting body and the emotions associated with specific movements” (p. 5502). Second, cognitive processing is intimately tied to both perception and action, so “appraising” an emotional signal, such as a vocal expression of Anger, may involve much more than simple information transfer between stored representations of emotion templates and encoded percepts of expressions.

Thus, recent theorizing about embodied cognition is generating new ideas and new data on how perception and action are related, both in experimental tasks and in neural architecture in the brain. Further, this body of work is demonstrating that many cognitive processes are not “cold” and “detached” but are intimately related to the perceiving/acting systems which humans use to adapt to their environments. Because part of that environment is social, it is not surprising that some of these perceiving/acting systems involve what Russell called “core affect,” namely, arousal and valence, both in ourselves and in our social partners.

9.6 Summary and Future Directions

Considerable agreement is evident in empirical studies of the vocal expression of Anger. Anger is one of the best identified emotions from sound alone, and Anger is usually associated with changes in fundamental frequency, intensity, and speech rate. With the notable exception of Sobin and Alpert

(1999), the fundamental frequency (what we typically hear as the pitch of the voice) increases during Anger, and Anger expressions tend to be more intense (i.e., louder to listeners) and faster. Further, when listeners are forced to choose between several alternative emotions as the one that they just heard, all studies show that Anger is identified at levels far above chance (typically calculated as the reciprocal of the number of alternative offered to participants).

Despite this fairly positive summary, important limitations of prior work, as well as new future directions, make vocal expressions of Anger a vital research area. As others have pointed out, previous work on vocal expressions of Anger has all employed variations on the same experimental paradigm. Examples of vocal expression are taken from recordings of actors and thus may represent shared, conventional ideas of what Anger *should* sound like. Further, these recordings are often pared down to the ones investigators find “best.” The forced-choice method of data collection provides limited information about how participants “hear” the similarities and differences among the emotion exemplars. The use of ratings scales, or even classic psychophysical techniques such as paired comparisons, would improve quantitative modeling of perceptual findings. Even working within the forced-choice paradigm, the issue of cross-cultural universality has been explored to a much lesser extent than it has for facial expressions. The few cross-cultural studies suggest that recognition of Anger decreases when cultural/linguistic lines are crossed, although recognition generally remains above chance. Finally, there have been demonstrable effects of listener and speaker differences (e.g., gender and individual differences), and no current theoretical model takes these into account very well.

Each of these criticisms of the “standard” paradigm has been noted before; some reviewers infer that the paradigm has outlived its usefulness (Bachorowski, 1999) and others call for slight modifications, including a wider range of acoustic measures and better standardization of the identity and strength of emotions portrayed (Juslin & Laukka, 2001). Indeed, different reviewers of the empirical literature find support for contradictory theoretical models. This state of affairs makes it seem likely that the models will remain entrenched, at least for the near future.

We have argued that methods follow partly from theory, and it seems clear that recent theoretical changes in psychology have begun to affect the emotions literature. In particular, the notion of embodied cognition suggests that “decoding” vocal expressions of Anger (and other emotions) is likely not a solely cognitive enterprise, or at least that cognition involves the neurophysiological and motor systems of the listener. The acoustic information may not carry all the “information” needed to decode a vocal expression. It may be that actions and responses engendered in the listener are crucial parts of recognizing emotions expressed by others. And perhaps these “listener effects” are one of the reasons that identification of Anger in vocal expressions has been less than perfect, even with carefully selected exemplars.

It is reasonable to ponder whether isolating the vocal expression of an underlying emotion, and then playing only the isolated vocal expression to listeners to judge, sets in motion a communication system that is fundamentally different from what takes place in everyday expression and understanding of emotions. We suggest, tentatively, that the answer is “yes,” it does, and that it may be useful to join the voice with the other parts of our perception/action system, to enhance our understanding of Anger and how it is communicated.

References

- American Psychological Association (2007). Controlling anger – before it controls you. <http://www.apa.org/topics/controlanger.html>
- Bachorowski, J. (1999). Vocal expression and perception of emotion. *Current directions in psychological science*, 8, 53–57.

- Bachorowski, J., & Owren, M. J. (2003). Sounds of emotion: Production and perception of affect-related vocal acoustics. In P. Ekman, J. J. Campos, R. J. Davidson, & F. B. M. de Waal (Eds.), *Emotions inside out: 130 years after Darwin's "The expression of the emotions in man and animals."* New York: New York Academy of Sciences.
- Banase, R., & Scherer, K. (1996). Acoustic profiles in vocal emotion expression. *Journal of Personality and Social Psychology*, 70(3), 614–636.
- Blair, R. J. R., Morris, J. S., Frith, C. D., Perrett, D. I., & Dolan, R. J. (1999). Dissociable neural responses to facial expressions of sadness and anger. *Brain*, 122, 883–893.
- Buck, R. (1999). *The biology of affects: A typology*. *Psychological Review*, 106, 301–336.
- Carr, L., Iacoboni, M., DuBeau, M., Mazziotta, J. C., & Lenzi, G. L. (2003). Neural mechanisms of empathy in humans: A relay from neural systems for imitation to limbic areas. *Proceedings of the national Academy of Sciences*, 100, 5497–5502.
- Denes, P. B., & Pinson, E. N. (1973). *The speech chain*. Garden City, New York: Anchor Press.
- Fairbanks, G., & Pronovost, W. (1939). An experimental study of the pitch characteristics of the voice during the expression of emotions. *Speech Monographs*, 6, 87–104.
- Galantucci, B., Fowler, C. A., & Turvey, M. T. (2006). The motor theory of speech perception reviewed. *Psychonomic Bulletin and Review*, 13, 361–377.
- James, W. (1894). What is an emotion? *Mind*, 19, 188–205.
- Johnstone, T., & Scherer, K. R. (2000). Vocal communication of emotion. In M. Lewis, J. M. Haviland-Jones (Eds.), *The handbook of emotions*. (2nd ed., pp. 220–235). New York: Guilford.
- Juslin, P. N., & Laukka, P. (2001). Impact of intended emotion intensity on cue utilization and decoding accuracy in vocal expression of emotion. *Emotion*, 1, 381–412.
- Juslin, P. N., & Laukka, P. (2003). Communication of emotions in vocal expression and music performance: Different channels, same code? *Psychological Bulletin*, 129, 770–814.
- Kent, R. D., & Read, W. (2002). *The acoustic analysis of speech*. (2nd ed.). San Diego: Singular.
- Kimbrell, T. A., George, M. S., Parekh, P. I., Ketter, T. A., Podell, D. M., Danielson, A. L., et al. (2001). Regional brain activity during transient self-induced anxiety and anger in healthy adults. *Biological Psychiatry*, 46, 454–465.
- Kruskal, J. P., & Wish, M. (1978). *Multidimensional scaling*. Newbury Park: Sage.
- Leinonen, L., Hiltunen, T., Linnankoski, I., & Laakso, M. (1997). Expression of emotional-motivational connotations with a one-word utterance. *Journal of the Acoustical Society of American*, 102(3), 1853–1863.
- Lieberman, A. M., & Mattingly, I. G. (1985). The motor theory of speech perception revised. *Cognition*, 21, 1–36.
- Murray, I. R., & Arnott, J. L. (1993). Toward a simulation of emotion in synthetic speech: A review of the literature on human vocal emotion. *Journal of the Acoustical Society of America*, 93, 1097–1108.
- Neumann R., & Strack, F. (2000). "Mood contagion": The automatic transfer of mood between persons. *Journal of Personality and Social Psychology*, 79(2), 211–223.
- Niedenthal, P. M., Brauer, M., Halberstadt, J. B., & Innes-Ker, A. H. (2001). When did her smile drop: Facial mimicry and the influences of emotional state on the detection of change in emotional expression. *Cognition and emotion*, 15, 853–864.
- Owren, M. J., & Bachorowski, J. (2007). Measuring emotion-related vocal acoustics. In J. A. Coan & J. J. B. Allen (Eds.), *Handbook of emotion elicitation and assessment*. New York: Oxford.
- Potegal, M. (2010). The temporal dynamics of anger: Phenomena, processes, and perplexities. In M. Potegal, G. Stemmler, & C. Spielberger (Eds.), *International handbook of anger: Constituent and concomitant biological, psychological, and social processes*. Secaucus, NJ: Springer.
- Prinz, W., & Hommel, B. (2002). *Common mechanisms in perception and action*. New York: Oxford.
- Ramirez, J. M. (2001). Moral approval of aggressive acts by urban students: A cross-national study on four continents. In J. M. Ramirez & D. S. Richardson (Eds.), *Cross-cultural approaches to research on aggression and reconciliation* (pp. 61–71). Huntington, New York: Nova Science Publishers.
- Russell, J. A. (2003). Core affect and the psychological construction of emotion. *Psychological Review*, 110(1), 145–172.
- Russell, J. A., Bachorowski, J., & Fernández-Dols, J. (2003). Facial and vocal expression of emotion. *Annual Review of Psychology*, 54, 329–349.
- Scherer, K. (1986). Vocal affect expression: A review and a model for future research. *Psychological Bulletin*, 99, 143–165.
- Scherer, K. R. (2000). Emotions as episodes of subsystem synchronization driven by nonlinear appraisal process. In M. D. Lewis & I. Granic (Eds.), *Emotion, development, and self-organization: Dynamic systems approaches to emotional development* (pp. 70–99). New York: Cambridge.
- Scherer, K. R. (2003). Vocal communication of emotion: A review of research paradigms. *Speech Communication*, 40, 227–256.

- Scherer, K. R., Banse, R., & Wallbott, H. G. (2001). Emotion inferences from vocal expression correlate across languages and cultures. *Journal of Cross-Cultural Psychology*, 32(1), 76–92.
- Scherer, K. R., Johnstone, T., & Klasmeyer, G. (2003). Vocal expression of emotion. In R. J. Davidson, K. R. Scherer, & H. Hill Goldsmith (Eds.), *Handbook of affective Sciences* (pp. 433–456). New York: Oxford University Press.
- Schieman, S. (2010). The sociological study of anger: Basic social patterns and contexts. In M. Potegal, G. Stemmler, & C. Spielberger (Eds.), *International handbook of anger: Constituent and concomitant biological, psychological, and social processes*. Secaucus, NJ: Springer.
- Siegman, A. W., & Snow, S. C. (1998). The outward expression of anger, the inward experience of anger and CVR: The role of vocal expression. *Journal of Behavioral Medicine*, 20, 29–45.
- Sobin, C., & Alpert, M. (1999). Emotion in speech: The acoustic attributes of fear, anger, sadness, and joy. *Journal of Psycholinguistic Research*, 28(4), 347–365.
- Van Bezooijen, R., Otto, S. A., & Heenan, T. A. (1983). Recognition of vocal expressions of emotion: A three-nation study to identify universal characteristics. *Journal of Cross-Cultural Psychology*, 14, 387–406.
- Whalen, P. J., Shin, L. M., & McInerney, S. C. (2001). A functional MRI study of human amygdala responses to facial expressions of fear versus anger. *Emotion*, 1, 70–83.
- Wranik, T., & Scherer, K. R. (2010). Why do I get angry: A componential appraisal approach. *International handbook of anger: Constituent and concomitant biological, psychological, and social processes*. Secaucus, NJ: Springer.

Chapter 10

Cross-Cultural Experience of Anger: A Psycholinguistic Analysis

Zoltán Kövecses

Abstract In this chapter, I will provide evidence for the embodied nature of the concept of anger and some of its metaphors from work in cognitive psychology. I will show that many unrelated languages and cultures do seem to share the generic-level metaphor: THE ANGRY PERSON IS A PRESSURIZED CONTAINER. This metaphor, I suggest, is motivated by the universal embodiment of anger. The pressurized container metaphor underlies the widespread conception that anger is a force that makes the angry person perform aggressive or violent actions. The actual physiology of anger provides much support for this conceptualization. At the same time, however, there is a considerable amount of variation in the counterparts of anger both cross-culturally and intraculturally. To account for some of this variation, a new, more nuanced view of embodiment will be proposed, where the major idea is that the embodiment of anger consists of multiple components, and cultures may choose which of these components they focus on. I will call this process of selecting one or several such components “experiential focus.” This idea helps us in part explain why, despite universal actual physiology, different cultures can have widely different understandings of their anger-like experiences.

This chapter examines some of the important dimensions of the cross-cultural experience of anger – especially the metaphors associated with it. As I have argued in a number of publications, metaphors play a major role in our understanding of abstract concepts in general and emotion concepts in particular (Kövecses, 1986, 1988, 1990, 2000a, 2005). Many metaphors have a constitutive role in the way we think about emotion concepts, including anger. Because of the importance of these metaphors in comparing the experience of anger across cultures, this chapter will not be a general survey of the cross-cultural study of anger. Instead, it proposes a new alternative to other approaches.

The relationship between metaphors and scientific theories of emotion has been elucidated in some previous publications (Kövecses, 1990, 2000a). In this chapter, I focus attention on the following specific issues relating to anger:

First, I will present some evidence that comes from cognitive psychology that the concept of anger is an embodied one. Second, I will show, again relying on work by cognitive psychologists, that the metaphors we use to understand the concept of anger have psychological reality, and they are not simply linguistic frills. Third, based on my previous research, I will suggest that the PRESSURIZED CONTAINER metaphor for anger is a near-universal metaphor. Fourth, I will show that in addition to the potential universality of several anger metaphors, they also exhibit a great deal of variation both

Z. Kövecses (✉)

Department of American Studies, Eötvös Loránd University, Budapest, Hungary
e-mail: zkovecses@ludens.elte.hu

cross-culturally and within cultures. Fifth, I will propose that it is the central metaphors of anger that give the various anger concepts much of their structure and content in different cultures. Sixth, in response to some recent challenges, I will offer a more nuanced view of the embodiment of anger. Seventh, I will briefly describe how embodiment, culture, and cognition jointly play a role in how anger is conceptualized.

10.1 The Embodiment of Anger

In cognitive linguistics, metaphor is a set of conceptual correspondences, or more technically, mappings, between two conceptual domains, a source and a target (Lakoff & Johnson, 1980; Kövecses, 2002). The correspondences between a source and a target domain make up a conceptual metaphor. It follows from this cognitive definition of metaphor that most conceptual metaphors will have linguistic instantiations in everyday language use (that is, they will be expressed by means of metaphorical linguistic expressions). Take, for instance, the conceptual metaphor ANGER IS A HOT FLUID IN A CONTAINER. This shows up in such metaphorical linguistic expressions as “*seethe* with anger,” “*boil* with anger,” “*simmer* down”. The basic mapping, or set of correspondences, that defines the conceptual metaphor that underlies these expressions includes, for example, “the physical container the angry person’s body.”

Psycholinguistic studies suggest that anger, like any abstract concept, is embodied. (Chapter 9 by Green et al., this book; on embodiment in general, see Gibbs, 2006.) In these studies, researchers ask people to think about and report on their embodied experiences concerning domains that are known, on the basis of linguistic evidence, to be used as source domains in conceptual metaphors, such as THE ANGRY PERSON IS A PRESSURIZED CONTAINER. However, in the experiments subjects are only asked about their experiences concerning the source domain; no mention is made of the target domain.

In a well-known series of experiments, Ray Gibbs (1992, 1994) asked his subjects about their embodied experiences concerning pressurized containers: What would cause the container to explode? Does the container explode on purpose or does it explode through no volition of its own? Does the explosion of the container occur in a gentle or a violent manner? People’s responses to these questions were remarkably similar. They agreed that the explosion happens as a result of internal pressure caused by the increase in the heat of the fluid inside the container; that the explosion happens unintentionally; and that the explosion happens in a violent manner. This way, the researcher generates a nonlinguistic profile of the embodied experience of pressurized containers that is one of the source domains of intense emotional states such as anger. With the help of such nonlinguistic profiles certain predictions can be made about people’s understanding of the target domains. This is possible only if in the course of comprehending a target domain in terms of a source, the source preserves its basic, generic-level profile or image-schematic structure. For example, it can be predicted that when the pressurized container as source domain is used for anger, the loss of control over anger that angry people often experience will be conceptualized as being caused by internal pressure, as being unintentional, as well as sudden and violent. These predictions, or hypotheses, concerning the conceptualization of anger proved to be the case in a variety of tasks. For example, when people understand idioms for anger (such as *blow your stack*, *flip your lid*, *hit the ceiling*), they infer that the loss of control that these idioms describe is due to some internal pressure, that it is unintentional, and that it occurs in an abrupt and violent manner. It is important to see that in these experiments the researcher attempts to discover people’s intuitions concerning their bodily experiences *before* any questions are asked concerning their judgments about linguistic expressions, their meaning or

their metaphorical status. It is such nonlinguistic profiles associated with source domains that are preserved for the structuring of target domains, thus providing them with conceptual structure and content. I suggest that many source domains, such as HOT FLUID IN A CONTAINER, or more generally, PRESSURIZED CONTAINER, for anger, are in the business of mapping such predetermined conceptual materials to the target – in this case, anger (see Kövecses, 2000b, 2002).

One of the remarkable features of metaphorical thought is that even our most basic target concepts can be construed in multiple ways. The metaphorical conceptual system is not monolithic – target concepts are not limited to a single source concept. Let us take as an example the abstract target concept of intensity. There is an experiential correlation between intensity and heat, in that when we perform a physical activity intensely or when we are in certain intense emotional states, the body produces more heat. We can now say that this correlation forms the basis of the conceptual metaphor INTENSITY IS HEAT. However, heat is not the only source domain for this target domain, as shown below:

INTENSITY IS HEAT (e.g., “There was *heated* debate about the issue”)

INTENSITY IS QUANTITY (e.g., “I care *a lot* about you”)

INTENSITY IS SPEED (e.g., “*sudden* growth in the economy,” “a *sluggish* economy”)

INTENSITY IS STRENGTH (OF PHYSICAL EFFECT) (e.g., “The country was *hit hard* by the flood”)

All these alternative conceptualizations of intensity are “primary” or “simple” metaphors that can jointly characterize particular “complex” metaphors (Grady, 1997a, b). When they do, we can think of them as providing very strong motivation for the selection of particular complex images. One case in point would be the complex conceptual metaphor ANGER IS A HOT FLUID IN CONTAINER. At least three of the four simple metaphors for intensity seem to be involved in this complex metaphor: HEAT, QUANTITY, and SPEED. If we lose our *cool*, we become very angry; anger *welling up* in someone indicates less intense anger than anger *coming over* or *overcoming* someone; and a person *flaring up* is more intensely angry than someone doing a *slow burn*. But maybe the fourth intensity metaphor also plays a role in this anger metaphor. For instance, an *outburst* of anger indicates very intense anger and also the forcefulness of the outbreak. Be that as it may, the point is that the extremely simple local metaphors that are based on basic correlations in human experience jointly apply to this complex metaphor and make it a very natural conceptual metaphor for anger.

10.2 The Psychological Reality of Anger Metaphors

Ray Gibbs and his associates were among the first to demonstrate that conceptual metaphors are real, i.e., they exist in our conceptual system and not just in our language (Gibbs & O’Brian, 1990; Gibbs, 1994). Participants first formed mental images of idioms (e.g., *blow your stack*, *flip your lid*, *hit the ceiling*) and were then asked a series of questions about their images (Gibbs & O’Brian, 1990). The images relating to idioms with roughly the same meaning (e.g., “getting angry”) were remarkably consistent across subjects. Participants made use of the image-schematic knowledge that was mentioned in the previous section. They said that in the case of idioms like *blow your stack*, the cause of losing control over anger is internal pressure, and that the loss of control is unintentional and violent. In other words, their responses were based on the source domain of pressurized container (like a hot fluid in a container). This means that in interpreting the idioms they relied on the conceptual metaphor ANGER IS A HOT FLUID IN A CONTAINER. If people’s knowledge were not

structured by such metaphorical mappings, there would be very little consistency in the images they get in connection with idioms with the same nonliteral meaning. What Gibbs and O'Brian showed was that people do indeed understand idioms relating to a given target domain (like anger) in terms of conceptual metaphors (such as ANGER IS A HOT FLUID IN A CONTAINER).

But understanding is not only a matter of long-term memory. It also involves the online, or real-time, understanding of language. One of the greatest challenges to the cognitive linguistic view of metaphor is the claim that conceptual metaphors play no role in online understanding. My specific claim is that we process metaphorical expressions online without (consciously or unconsciously) evoking or relying on metaphorical mappings.

Gibbs and his associates (Gibbs, Bogdonovich, Sykes, & Barr, 1997) took up the challenge; in particular, they wanted to see how people immediately comprehend metaphorical idioms based on ANGER IS A HOT FLUID IN A CONTAINER, such as *blow one's stack*. Participants read stories ending with idioms, such as this, and then quickly gave lexical decision responses to letter strings that were presented to them visually. The letter strings had to do with either the conceptual metaphor underlying the idioms or they were unrelated to them. For example, a related letter string was "heat," and an unrelated one was "lead." People responded faster to the lexical decision questions after they were presented with a related letter string than when they were with an unrelated one, such as "lead." Findings in a variety of tasks were consistent. All of this research shows that people do make some use of conceptual metaphors when they comprehend metaphorical expressions in real time.

10.3 The Universality of the PRESSURIZED CONTAINER Metaphor for Anger

The ANGER IS A HOT FLUID IN A CONTAINER (and its generic version THE ANGRY PERSON IS A PRESSURIZED CONTAINER) metaphor was first studied by Lakoff and Kövecses in English (Kövecses, 1986; Lakoff, 1987; Lakoff & Kövecses, 1987), and then by a number of researchers in both related and unrelated languages including Chinese (King, 1989; Yu, 1995, 1998), Japanese (Matsuki, 1995), Hungarian (Bokor, 1997), Wolof (Munro, 1991), Zulu (Taylor & Mbense, 1998), Polish (Micholajczuk, 1998), and some others (for a summary, see Kövecses, 2000a). In addition to the expressions given above, here are some linguistic metaphors that express this conceptual metaphor in English: "Let him *stew*," "You make my blood *boil*," "He *blew his top*," "Simmer down!"

In all of these languages, a CONTAINER metaphor was found for anger, and the container was found pressurized, either with or without heat. The correspondences, or mappings, of the PRESSURIZED CONTAINER metaphor for anger include

- the container with some substance or objects → the angry person's body
- the substance or objects in the container → the anger
- the pressure of the substance or objects on the container → the force of the anger on the angry person
- the cause of the pressure → the cause of the anger force
- keeping the substance or objects inside the container → controlling the anger
- the substance or objects going out of the container → the expression of the anger

As we will see later, these correspondences play a key role in the constitution of the concept of anger. Through its detailed mappings, the metaphor provides a coherent structure for the various "anger-like" concepts in different languages.

Table 10.1 Entailments of the ANGER IS A HOT FLUID IN A CONTAINER METAPHOR

Metaphorical entailments	Corresponding linguistic examples
WHEN THE INTENSITY OF ANGER INCREASES, THE FLUID RISES:	His pent-up anger <i>welled up</i> inside him
INTENSE ANGER PRODUCES STEAM:	Billy's just <i>blowing off steam</i>
INTENSE ANGER PRODUCES PRESSURE ON THE CONTAINER:	He was <i>bursting with</i> anger
WHEN ANGER BECOMES TOO INTENSE, THE PERSON EXPLODES:	When I told him, he just <i>exploded</i>
WHEN A PERSON EXPLODES, PARTS OF HIM GO UP IN THE AIR:	I <i>blew my stack</i>
WHEN A PERSON EXPLODES, WHAT WAS INSIDE HIM COMES OUT:	His anger finally <i>came out</i>

The PRESSURIZED CONTAINER metaphor gives rise to a series of metaphorical entailments. In English, these are given in Table 10.1

Many of these entailments are shared by several of the languages that were studied. Hungarian, Japanese, and Chinese have most of them, but the other languages have them as well to varying degrees, as the evidence of linguistic expressions makes it clear.

This is an extraordinary situation. How can speakers of such diverse languages as Chinese, Hungarian, Zulu (spoken in South Africa), Wolof (spoken in West Africa), and possibly many others around the world, have conceptualized an “anger-like” experience in such remarkably similar ways? First of all, we should look at the anger-related metonymies in diverse languages. Metonymy is a cognitive process in which an entity in a domain or frame provides mental access to another entity within the same domain or frame (see Kövecses & Radden, 1998). Many of the languages for which we have data share several important conceptual metonymies that include

BODY HEAT STANDS FOR ANGER

INTERNAL PRESSURE STANDS FOR ANGER

REDNESS IN FACE AND NECK AREA STANDS FOR ANGER

In line with the above definition of metonymy, the mention of body heat, internal pressure, and redness in the face and neck area can be used to provide mental access to anger. In general, the metonymies tend to describe the physiological, behavioral, and expressive reactions in the case of emotion.

Actually, we seem to have some (though not conclusive) nonlinguistic evidence for the universality and the anger specificity of such physiological responses. Paul Ekman, Levenson, and their colleagues (Ekman, Levenson, & Friesen, 1983; Levenson, Ekman, & Friesen, 1990; Levenson, Carstensen, Friesen, & Ekman, 1991) provide evidence that anger is indeed associated with objectively measurable bodily changes such as increase in skin temperature, blood pressure, pulse rate, and more intense respiration; other emotions, like fear and sadness, go together with a different set of physiological reactions. These studies were conducted with American subjects only. However, Levenson and his colleagues extended their research cross-culturally and found that emotion-specific ANS (autonomic nervous system) activity was the same in Americans and the Minangkabau of West Sumatra (Levenson, Ekman, Heider, & Friesen, 1992). For example, skin temperature and pulse rate rise in anger in both American and Minangkabau subjects. These findings give us reason to believe that the actual physiology might be universal. The universality of actual physiology might be seen as leading to the similarities (though not equivalence) in conceptualized physiology (i.e., the conceptual metonymies), which might then lead to the similarity (though again not equivalence) in the metaphorical conceptualization of anger and its counterparts (i.e., the PRESSURIZED CONTAINER metaphor). (On this issue, see also Chapter 7 by Stemmler, this volume.)

The conceptual metonymies mentioned above capture people's folk theory of some of the physiology of anger. English, Hungarian, Japanese, Zulu, Polish, Wolof, and, to some degree, Chinese as well seem to share the notion of an increase in body heat in anger, and they also talk about it metonymically. The notion of subjective body heat, perhaps together with the idea of the felt warmth of blood, seems to be the cognitive basis for the heat component of the English, Hungarian, Japanese, and Wolof CONTAINER metaphors. The fact that Chinese does not have a large number of metonymies associated with body heat may be responsible for the Chinese CONTAINER metaphor *not* involving a *hot* fluid or gas.

Internal pressure is present in English, Chinese, Japanese, Hungarian, Polish, and Zulu. The physiological response "redness in the face and neck area" can be taken to be the result of both body heat and internal pressure. This response seems to characterize English, Chinese, Japanese, Hungarian, Polish, and Zulu. The Wolof word *boy* "to be red hot (of charcoal)" also means "to be really angry."

My proposal here was that conceptualized physiology (i.e., the conceptual metonymies) provides the cognitive motivation for people to conceptualize the angry person metaphorically as a PRESSURIZED CONTAINER (Kövecses, 2000a). The PRESSURIZED CONTAINER metaphor is well motivated by the conceptual metonymies, in that it is mainly based on the notions of heat and pressure. The metonymies make this particular metaphorical conceptualization natural for people. If conceptualized physiological responses include an increase in internal pressure as a major response in a given culture, people in this culture will find the use of the PRESSURIZED CONTAINER metaphor natural.

Without the constraining effect of universal embodiment, it is difficult to see how such a surprisingly uniform category (of a variety of PRESSURIZED CONTAINER metaphors) could have emerged for the conceptualization of anger and its counterparts in very different languages and cultures. But these changes in physiology in anger may overlap with physiological changes in other emotions. This is why happiness can also have the PRESSURIZED CONTAINER metaphor (as indicated by examples such as "*bursting* with joy"), although without the strong heat component that characterizes anger in English-speaking subjects. Thus, a more general conceptual metaphor that could account for such cases would be A PERSON IN AN INTENSE EMOTIONAL STATE IS A PRESSURIZED CONTAINER. The main meaning focus of the metaphor is "difficulty in controlling a process," which in turn derives from the mapping "difficulty in controlling a (n emotional) process → difficulty in keeping a substance in a pressurized container." It is this mapping that characterizes the PRESSURIZED CONTAINER metaphor in its various applications to emotions other than anger and in languages other than English. For example, Palmer, Bennett, and Stacey (1999) point out that the metaphor is applied to the emotion concepts of shame and grief in Tagalog.

It is important to note that the "same" bodily phenomenon may be interpreted differently in different cultures and that activities of the body (and the body itself) are often "construed" differentially in terms of local cultural knowledge (see, for example, Csordas, 1994; Gibbs, 1999). In the present context, physically identical bodily activities can receive different meanings in two different cultures (or subcultures). And yet, it seems that the kinds of bodily experiences that form the basis of many conceptual metaphors (such as the PRESSURIZED CONTAINER metaphor for anger and some other intense emotions) can and do exist independently of any cultural interpretation – either conscious or unconscious. They are products of the kinds of physical bodies that we have. However, this is not to say that these products of the body cannot be shaped by local cultural knowledge.

10.4 Cross-Cultural and Intracultural Variation in Anger

Although THE ANGRY PERSON IS A PRESSURIZED CONTAINER metaphor seems to be a widespread, even near-universal metaphor, it can occur in somewhat different forms in different cultures. Other metaphors for anger may reveal the same kind of variation. In the present section, I will look at some

of the types of variation that can occur in the various aspects, or components, of this key metaphor for anger.

10.4.1 *Specificity and Congruence of the Source*

Consider all the specific-level manifestations of the generic-level PRESSURIZED CONTAINER metaphor for anger. In American English, the dangerous PRESSURIZED CONTAINER is specified as A HOT FLUID IN A CONTAINER at a lower level of conceptual organization or abstraction, and at a very low level of specificity it can be further specified as a VOLCANO, a FUSE, an EXPLOSIVE, a COW GIVING BIRTH, and so on. These latter, highly specific source domains are likely to be language specific, much more so than either the HOT FLUID metaphor or the PRESSURIZED CONTAINER metaphor, which is a potentially universal conceptual metaphor. In another case, Hungarian shares with English the conceptual metaphors THE BODY IS A CONTAINER FOR THE EMOTIONS and ANGER IS FIRE. The body and the fire inside it are commonly elaborated in Hungarian as a PIPE, where there is a burning substance inside a container. This conceptual elaboration seems to be unique to Hungarian. Hungarians also tend to use the more specific container of the HEAD (with the brain inside) for the general BODY CONTAINER in English in talking about anger, and a large number of Hungarian expressions indicate how anger can affect the head and the brain.

10.4.2 *Entailments*

Let us now compare some of the entailments of some of the anger metaphors in English and Zulu. The analysis of the Zulu anger metaphors is based on Taylor and Mbense's (1998) work on Zulu anger.

Both English and Zulu have FIRE as a source domain for anger, but speakers of Zulu make use of inferences (or entailments) concerning the metaphor in a way in which speakers of English do not. In Zulu one can *extinguish* somebody's anger *by pouring water* on them. This possible metaphorical entailment is not picked up by the English FIRE metaphor in the form of conventionalized linguistic expressions. What comes closest to the Zulu expression in English is the stylistically limited (literary–religious) use of the verb *quench* (see Chapter 22 by Potegal, this volume). However, this does not represent widespread, everyday usage, as the Zulu expression does. In this sense, this entailment of the FIRE source domain that applies to anger in Zulu does not exist in English in a conventional form.

The cultural, or cognitive, model of anger has desire (to harm) as a component in both English and Zulu. This can be found as a submapping (or submetaphor) of the ANGER IS A WILD ANIMAL metaphor: DESIRE IS HUNGER. This submetaphor appears to exist in Zulu as well, but it seems to have unique entailments for speakers of Zulu. We can interpret Taylor and Mbense's (1998) description of Zulu anger in such a way as to suggest that in Zulu an angry person's appetite can be so voracious that he eats food that is not even prepared or he does not even separate edible from inedible food. This aspect of the metaphor is obviously missing from English, at least as judged by the English conventionalized linguistic expressions.

In both English and Zulu, anger can be comprehended as A NATURAL FORCE. But speakers of Zulu go much further in making use of the entailment potential of this metaphor than speakers of English. In Zulu one can say of an angry person that *the sky became dark with thunderclouds*, *the sky* (= lightning) *almost singed us*, or *why did he blow a gale*? These entailments do not exist in English in conventionalized form, but speakers of English may well understand them given the shared conceptual metaphor.

10.4.3 Linguistic Expressions for the Same Conceptual Metaphor

Most of the differences between conceptual metaphors in any two languages will occur at the level of linguistic expressions. The generic conceptual metaphors may be shared, even some of the specific conceptual metaphors may be shared, but we can expect a great deal of variation in the exact phrasing of the linguistic metaphors that express a particular anger-related conceptual metaphor. Here are some examples in Tunisian Arabic (Maalej, 2003):

ma-bqaaš 3and-i wayn ydur ir-riiH.
No exist with me where circulate the wind
There is no more room for air to circulate inside me.
I could barely keep it in anymore.

Talla3-l-i id-damm l-raaS-i.
[He]lift-past to me the blood to head my
He lifted blood up to my head.
I was flushed with anger.

Haraq-l-i muxx-i.
[He]burn-past to me brain my
He burnt my brain to me.
He caused my blood to burn.

It is probably only Arabic speakers who express these figurative meanings related to anger in the particular ways they do: as wind circulating inside the person, as blood being lifted to one's head, and as someone burning your brain. The meanings can be translated into English, and they sound familiar. However, the actual phrasing in Tunisian Arabic is radically different from what we find in English or Hungarian. This is so despite the fact that shared conceptual metaphors are utilized: ANGER IS PRESSURE INSIDE THE PERSON in the first two examples and ANGER IS HEAT in the third. What this situation suggests is that we have the same figurative meaning expressed by words whose literal meaning differs markedly from that of English words used for the same purpose and which are expressions of the same conceptual metaphors.

10.4.4 Degree of Linguistic Elaboration

A shared conceptual metaphor can be elaborated differently in different languages/varieties. Elaboration here means that a given mapping or metaphorical entailment gives rise to a larger or fewer number of linguistic expressions in two languages/varieties. For example, the metaphor ANGER IS A HOT FLUID IN A CONTAINER has, among others, the following mappings and entailments in English: "pressure inside the container → difficult-to-control anger in the person," "the container exploding → the person losing control over anger," and "when the person-container explodes, parts of him go up in the air." These aspects of the metaphor are highly elaborated in American English but less so in Spanish (Barcelona, 2001). In American English, you can *have a cow*, *blow a fuse*, *blow a gasket* and *flip your lid*, *blow your top*, *blow your stack*, *fly off the handle*, but these expressions do not seem to have equally conventionalized counterparts in Spanish. This is not to say that Spanish has no means of talking about this aspect of anger; rather, the claim is that it has a much less elaborated repertory of conventionalized linguistic expressions to do it. For example, in Spanish one can say *Se voló la olla*, "His kettle (i.e., his head) blew up," which, according to Barcelona (2001), is not limited to anger.

10.4.5 *Variation in the Understanding of Anger in the Same Culture Through Time: The United States*

Was anger always predominantly conceptualized in the United States as it is today, that is, in terms of the HOT FLUID IN A PRESSURIZED CONTAINER metaphor? This is a difficult question for a cognitive linguist to answer, but, fortunately, social historians of American culture come to our rescue. Peter Stearns (1994) offers an excellent history of emotions in America in his book *American Cool*. Stearns' study shows that the conceptualization of anger, and of emotions in general, underwent important changes from the eighteenth to the nineteenth century. According to Stearns (1994, pp. 66–67), before the nineteenth century, the concept of anger was (and emotions in general were) primarily understood metonymically, rather than metaphorically. There was a great deal of emphasis on what actually happens to the body while in intense emotional states, such as hot blood for anger and cold sweats for fear. This emphasis on “embodiment” was replaced by metaphoric thinking about anger in the course of the nineteenth century, which resulted in viewing anger in humoral terms, that is, in terms of the body as a container with fluids in it. This conception comes close to, although is still not the same as, the now dominant HOT FLUID metaphor. In order for this particular metaphor to emerge, certain changes had to occur in the general social and cultural setting. In sum, what we can see here is a gradual shift from metonymic to metaphoric understanding, and, later on, from one metaphoric understanding to another.

As Stearns (1994) notes in connection with Victorian emotionology, anger was not a permissible emotion in the home, but, for men, it was actually encouraged at the workplace and in the world of politics. Women were supposed to be “anger-free,” and men, while calm at home, were expected to make good use of their anger for purposes of competition with others and for the sake of certain moral ends. But why did this “channeled anger” give way to the ideal of “anger-free” people or to the ideal of suppressing anger under all circumstances, as presupposed by the now dominant PRESSURIZED CONTAINER metaphor? Why did anger become a completely negative emotion? There were a variety of specific reasons, as Stearns argues, including the following:

New levels of concern about anger and aggression followed in part from perceptions of heightened crime, including juvenile delinquency, and the Results of untrammelled aggression in Nazism and then renewed world war. It was difficult, in this context, to view channeled anger as a safe or even useful emotional motivation (p. 195).

As a result, the attacks on any form of anger, which started around the 1920s, continued throughout the depression period and the Second World War, leading to a global rejection of the emotion by the 1960s in mainstream culture. The new metaphoric image that became prevalent was that of the “pressure cooker waiting to explode,” that is, the metaphor that we call ANGER IS A HOT FLUID IN A PRESSURIZED CONTAINER today. This was a fully mechanical metaphor that depicted anger as something completely independent of the rational self, the angry person as incapable of any rational judgment, and the resulting angry behavior as extremely dangerous. The process (that started in the eighteenth century) of the separation of the emotion from the self and the body, i.e., the “mechanization” of anger, was now completed.

It is important to see that this brief history concerns the cultural model, the folk theory, of anger in the United States – and not its expert or scientific theories. Experts, like psychologists, can and do talk about the positive aspects of anger. As a matter of fact, what kind of anger is considered prototypical in a given culture seems to be largely a matter of what the cultural context (see below). The view of anger as a potentially positive, constructive force is also present today in many cultures, including the United States, though not as the prototype of anger.

We might add to Stearns' causes of the change in the conceptualization of anger, as Michael Potegal (personal communication) suggested, that the change was also due to the increasing salience of the machinery of the industrial revolution, e.g., the steam engine, in people's lives.

10.5 The Concept of Anger and Its Central Metaphors in Different Languages and Cultures

Lakoff and Kövecses (1987) characterized the naive, or folk, understanding of anger in English as a prototypical cognitive, or cultural, model. They suggested the following model based on linguistic evidence in American English:

1. Offending event
 - Wrongdoer offends self.
 - Wrongdoer is at fault.
 - The offending event displeases self.
 - The intensity of the offense outweighs the intensity of the retribution (which equals zero at this point), thus creating an imbalance.
 - The offense causes anger to come into existence.
2. Anger
 - Anger exists.
 - Self experiences physiological effects (heat, pressure, agitation).
 - Anger exerts force on the self to attempt an act of retribution.
3. Attempt to control anger
 - Self exerts a counterforce in an attempt to control anger.
4. Loss of control
 - The intensity of anger goes above the limit.
 - Anger takes control of self.
 - Self exhibits angry behavior (loss of judgment, aggressive actions).
 - There is damage to self.
 - There is danger to the target of anger, in this case, the wrongdoer.
5. Retribution
 - Self performs retributive act against wrongdoer (this is usually angry behavior).
 - The intensity of retribution balances the intensity of offense.
 - The intensity of anger drops to zero.
 - Anger ceases to exist.

The main idea here was that the metaphors and metonymies associated with anger converge on and constitute the model, with the different metaphors and metonymies mapping onto different parts of the model.

Native speakers of Hungarian seem to have very much the same cultural model of anger (*düh* in Hungarian). The *but*-test that Lakoff and Kövecses (1987) used to ascertain the validity of the model for English yields the same Results for speakers of Hungarian as it does for speakers of English. For example, the sentence "He was angry, but he didn't lose control" and its Hungarian equivalent sound more natural than the sentence "He was very angry, but he lost control" in both languages. This is because the conjunction "*but*" is used to counter expectations. In this case, the expectation dictated by the prototypical model would be that once we are very angry (Stage 2), we tend to lose control (Stage 4). In other words, the applicability of the *but*-test indicates deviation from the prototypical cultural model. Since it indicates the same kinds of deviations in the two languages, it also shows that the underlying prototypical cultural models have a similar overall structure. (On using the *but*-test in psycholinguistic experiments concerning anger, see Gibbs, 1994.)

In the characterization of Japanese *ikari* (and, less typically, also *hara*), Matsuki (1995) notes in connection with the model found in American English: “The scenario applies to Japanese anger, although Stage 3 is more elaborate than in English” (p. 145). In the Japanese conception, the control aspect of *ikari* is more elaborate because anger first appears in *hara*, then it goes up to *mune*, and finally to *atama*. As Matsuki points out, *hara* is a container (the stomach/bowels area) and, metonymically (CONTAINER FOR CONTENT), can also be the emotion itself. *Mune* is the chest and *atama* is the head. If anger reaches *atama*, the angry person is unable to control anger.

King (1989) suggests that there are two prototypical cognitive models operating in Chinese:

1. Offending Event
 - Wrongdoer offends self.
 - The offending event displeases self.
 - The offense causes an imbalance in the body.
2. Anger
 - Anger exists.
 - Self experiences physiological effects (heat, pressure, agitation).
3. Attempt to control anger
 - Self exerts a counterforce in an attempt to control anger.
4. Release of anger
 - Self releases anger by exhibiting angry behavior.
5. Restoration of equilibrium
 - The amount of discharged anger balances the excess in the body.
 - The imbalance disappears and equilibrium is restored.
 - The other model differs from the one above in stages 4 and 5:
4. Diversion
 - The force of anger is diverted to various parts of the body.
 - Self exhibits somatic effects (headaches, stomachaches, etc.)
5. Compensating event
 - The compensating event pleases the self (this is usually sympathetic behavior directed at self).
 - The intensity of compensation balances the intensity of the offense.
 - The somatic effects of anger disappear.
 - Anger ceases to exist.

In addition to the several differences, we find several things in common to these models. They all seem to be composed of several successive stages and they all seem to have an ontological, a causal, and an expressive aspect. Based on the characterizations given above, the following general structure of the respective emotion concepts (*anger*, *düh*, *ikari/hara*, and *nu*) can be identified.

The prototypical cognitive models have an *ontological* part that gives us an idea of the ontological status and nature of anger, that is, the kind of thing/event it is. In all four languages anger, or its counterpart, is a force inside the person that can exert pressure on him or her. The ontological part also includes some physiological processes associated with the respective emotion. It is the ontological part of the model that constitutes the second stage of the cognitive model or scenario as a whole.

The first stage in the model corresponds to the *causal* part. This presents anger and its counterparts as an emotion that is caused, or produced, by a certain situation.

Still another part of the model is concerned with the *expressive* component; that is, the ways in which anger, or its counterpart is expressed in the different cultures. The cognitive models tell us that all four cultures conceive of anger as something that is somehow expressed.

Finally, the expressive component is preceded by a *control* component that is manifested as two separate stages of the model: attempt at controlling expression and loss of control over expression.

Thus, the resulting five-stage model for the four cultures seems to be the following:

- (1) cause → (2) existence of anger, or its counterpart (in the form of a force) → (3) attempt at control → (4) loss of control → (5) expression

(Here, the arrow → indicates temporal succession and causal sequence). Since expression and control are closely linked (i.e., at issue is the control of expression), it is possible to conceive of the two as a single aspect and refer to them as the expression component of the model, yielding the highly schematic model:

cause → existence of emotion (as forceful entity) → expression.

This then seems to be the *basic skeletal structure* that all four cultures share in their folk understanding.

But how can metaphors create such a model? My suggestion is that this happens by means of the set of mappings that characterize conceptual metaphors. Some metaphors play a central role in defining a particular model for a concept. In the case of anger, the central metaphor that “lends” much of the structure to the model of anger in a variety of cultures is that of PRESSURIZED CONTAINER. The particular structure that anger and other emotion concepts share is the “cause–existence of emotion–expression” schema. This is defined, in large part, by the PRESSURIZED CONTAINER metaphor that is characterized by the mappings we saw above. I believe that these are the mappings that play a constitutive role in the construction of the basic structure of the folk understandings of anger and its counterparts in different cultures. Without these mappings (i.e., imposing the schematic structure of how the force of a fluid or gas behaves in a container onto anger), it is difficult to see how anger and its counterparts could have acquired the structure they seem to possess. Without the PRESSURIZED CONTAINER metaphor, the “cause–emotion force–involuntary expression” structure would remain a mystery.

In the view presented here, the conceptual metaphors and metonymies contribute actively to the structure and content of the prototypical cultural models. In Zulu, the chief conceptual metaphor that provides the skeletal structure for anger is a version of the PRESSURIZED CONTAINER metaphor: ANGER IS IN THE HEART (Taylor & Mbense, 1998; Kövecses, 2000a). However, just like in English, additional metaphors focus on particular aspects of this generic structure. In the case of Zulu anger, two metaphors are especially important for the “expression” part of the basic model, which specifies the nature and intensity of angry behavior. Speakers of Zulu elaborate on two metaphors that speakers of English do not or do to a much smaller degree: ANGER (DESIRE) IS HUNGER and ANGER IS A NATURAL FORCE (Taylor & Mbense, 1998). If the metaphor DESIRE IS HUNGER is elaborated as voracious appetite that devours everything indiscriminately and NATURAL FORCE as a force that destroys everything, as is the case in Zulu, then this will probably influence the cultural model of anger, as is indeed the case according to Taylor and Mbense. Instead of venting their anger on a specific target (in English, the person who offended you), Zulu people appear to respond in a less clearly directed way and behave aggressively toward everyone indiscriminately. This is not to say that English cannot have this response or that Zulu cannot have the directed response; rather, the two languages seem to differ in what they consider the prototypical cultural model for the concept.

The major claim I am making here is that systematic links take us from (possibly universal) actual physiology of anger through conceptualized metonymy and metaphor to cultural models. In the process, the broader cultural contexts also play a crucial role, in that they fill out the details left open in the schematic basic structure. In other words, I believe that we can offer a satisfactory explanation of the emergence of cultural models if we take into account the possibly universal experiential

basis of most of our abstract concepts, the conceptualization of this experiential basis by means of conceptual metonymies, the conceptual metaphors that often derive from these metonymies, and the broader cultural context. The central conceptual metaphor in the case of anger is the PRESSURIZED CONTAINER metaphor (and the generic FORCE metaphor for the emotions in general; see Kövecses, 2000a), but other domains would be structured by other central metaphors. We should of course not imagine the process of the emergence of cultural models in sequential steps, going from experiential basis to cultural model. A probably more adequate way of thinking about it would be to say that the components outlined here are all at work at the same time, mutually influencing each other. In the course of this joint evolution, the conceptualized experiential basis (often appearing as conceptual metonymies) and the emerging conceptual metaphor contribute to the basic schematic structure of the cultural model, while the simultaneously present cultural context fleshes out the details of the schema.

10.5.1 *The Role of Cultural Context in Variation*

By cultural context I simply mean the broader context that a culture or subculture provides for the understanding of any of its concepts, including all the (sub)culturally unique and salient concepts and values that characterize particular (sub)cultures – together with the governing principles of a given culture or subculture. The governing principles and key concepts have special importance in (metaphorical) conceptualization because they permeate several general domains of experience for a culture or cultural group.

To demonstrate the effect of these differences on metaphor, let us first consider in some detail the near-universal PRESSURIZED CONTAINER metaphor for anger in a variety of cultures. We have seen above that at a generic level, this metaphor is very similar across many cultures. However, I also pointed out that at a specific level we can notice important differences in the metaphor across certain cultures. How do the differences arise?

Geeraerts and Grondelaers (1995) note that in the Euro-American tradition (including Hungary), it is the classical–medieval notion of the *four humors* from which the Euro-American conceptualization of anger (as well as that of emotion in general) derived. (We may also note that the Hungarian concept of *düh* also comes from the same source.) But they also note that the application of the humoral doctrine is not limited to anger or the emotions. The humoral view maintains that the four fluids (phlegm, black bile, yellow bile, and blood) regulate the vital processes of the human body. They were also believed to determine personality types (such as sanguine, melancholy) and account for a number of medical problems, together with cures for them (like blood-letting). Obviously, then, the use of the humoral view as a form of cultural explanation extends far beyond anger and the emotions. In addition to being an account of emotional phenomena, it was also used to explain a variety of issues in physiology, psychology, and medicine. In other words, the humoral view was a key component of the classical–medieval cultural context and it exerted a major impact on the emergence of the European conception of anger as a fluid in a pressurized container.

In Japan, as Matsuki (1995) tells us, there seems to exist a culturally distinct set of concepts that is built around the concept of *hara* (meaning both the belly/stomach area and anger). Truth, real intentions, and the real self (called *honne*) constitute the content of *hara*. The term *honne* is contrasted with *tatemae* or one's social face. Thus when a Japanese person keeps his anger (*ikari*) under control, he or she is hiding his or her private, truthful, innermost self and displaying a social face that is called for in the situation by accepted standards of behavior. The notion of *hara* has greatly influenced the Japanese conception of anger over the ages.

King (1989) and Yu (1995, 1998) suggest that the Chinese concept of *nu* (corresponding to anger) is bound up with the notion of *qi*, that is, the energy that flows through the body. *Qi* in turn is embedded in not only the psychological (i.e., emotional) but also the philosophical and medical discourse of Chinese culture and civilization. The notion and the workings of *qi* are predicated on the belief that the human body is a homeostatic organism, the belief on which traditional Chinese medicine is based. And the conception of the body as a homeostatic organism seems to derive from the more general philosophical view that the universe operates with two complementary forces, *yin* and *yang*, which must be in balance to maintain the harmony of the universe. Similarly, when *qi* rises in the body, there is anger (*nu*), and when it subsides and there is balance again, there is harmony and emotional calm. Without the concept of “qi,” it would be difficult to imagine the view of anger in Chinese culture.

Thus the four emotion concepts, *anger* in English, *düh* in Hungarian (the two representing European culture), *ikari* in Japanese, and *nu* in Chinese, are in part explained in the respective cultures by the culture-specific concepts of the *four humors*, *hara*, and *qi*. What accounts for the distinctiveness of the culture-specific concepts is the fact that, as we have just seen, the culture-specific concepts that are evoked to explain the emotion concepts are embedded in very different systems of cultural concepts and propositions. It appears then that the broader cultural contexts that operate with culture-specific key concepts account for many of the specific-level differences among the four emotion concepts and the PRESSURIZED CONTAINER metaphor.

10.6 A More Nuanced View of the Embodiment of Anger: “Experiential Focus”

It is a fundamental claim of the theory presented here that in many cases human beings share a great deal of bodily experience on the basis of which they can build universal metaphors. The question that inevitably arises is this: Is this universal bodily basis utilized in the same way across languages and cultures or even varieties? In light of the available evidence it seems that the answer is no. The universal bodily basis on which universal metaphors *could* be built is *not* utilized in the same way or to the same extent in different languages and varieties of languages. The notion that I would like to offer to get clear about this issue is that of “differential experiential focus.” What this means is that different peoples may be attuned to different aspects of their bodily functioning in relation to a target domain, or that they can ignore or downplay certain aspects of their bodily functioning as regards the metaphorical conceptualization of a particular target domain.

The conceptualization of anger in English and Chinese offers a good example to prove the point. As studies of the physiology of anger across several unrelated cultures suggest that increase in skin temperature and blood pressure may be universal physiological correlates of anger. This accounts for the ANGER IS HEAT metaphor in English and in many other languages. However, King’s (1989) and Yu’s (1995, 1998) works suggest that the conceptualization of anger in terms of heat is much less prevalent in Chinese than it is in English. In Chinese, the major metaphors of anger seem to be based on pressure, not on pressure *and* heat. This indicates that speakers of Chinese have relied on a different aspect of their physiology in the metaphorical conceptualization of anger than speakers of English. (Assuming that their physiological response to anger does not differ from that of English speakers.) The major point is that in many cases the universality of experiential basis does not necessarily lead to universally equivalent conceptualization at the specific level of hot fluids, in the case of anger. But, as we saw, at a generic level near-universality does occur.

Another example of how different cultures utilize a presumably universal bodily basis in anger is offered by Michelle Rosaldo in her description of Ilongot anger (Rosaldo, 1980). The Ilongot are a former headhunting tribe living in Northern Luzon, Philippines. For young Ilongot men, anger, *liget*, is a highly energized state that they need in order to successfully accomplish their headhunting raids. In Rosaldo's words "The *liget* that Ilongots associate with youthful prowess and, for them, with the universal agitation that makes young men want to kill, takes on reality and significance because it is bound up not in mystery or cosmology, but in three forms of relation central to Ilongot social life" (Rosaldo, 1980, p. 138). Indeed, Rosaldo glosses the Ilongot term for anger as "energy/anger." This suggests that for the Ilongot anger (*liget*) figures as a generalized state of arousal that can sufficiently motivate their actions. They think of their anger also as hot but, most importantly, as an agitated and energized state that makes them want to go out and take heads. Clearly, this is, for us, a surprisingly different way of building on our presumably universal bodily experience in conceptualizing anger.

As a matter of fact, the conceptualization of anger in terms of heat has not always been the case even in English. Carolyn Gevaert (2001, 2005) found on the basis of a variety of historical corpora that heat-related words for anger fluctuate a great deal in the Old English and Middle English period. According to Gevaert, her data indicate that the conceptualization of anger in terms of heat is not a permanent and ever-present feature of the concept of anger in English. She suggests that if her findings are correct, they invalidate, or disprove, the embodiment hypothesis. Her reasoning is that if the idea that people's concepts of anger are embodied in universal (physiological) experience, then people's conceptualization of anger cannot change over time.

I would like to suggest that universal physiology provides only a *potential* basis for metaphorical conceptualization – without mechanically constraining what the specific metaphors for anger will be. I believe it is best to replace a mechanical notion of embodiment and rely on the new notion of "differential experiential focus" (see Kövecses, 2005).

Heat was a major component in the concept of anger between AD 850 and 950, and then after a long decline it began to increase again at around 1400 – possibly as a result of the emergence of the humoral view of emotions in Europe (see Gevaert, 2001, 2005; Geeraerts & Grondelaers, 1995). We can observe the same kind of fluctuation in the use of the domain of "swell," which I take to be akin to what we call the "pressure" component in the conceptualization of anger today. Pressure was a major part of the conceptualization of anger until around 1300, but then it began to decline, only to emerge strongly again, together with heat, in the form of the HOT FLUID IN A CONTAINER metaphor centuries later. The point is that we should not expect any of the *conceptualized* responses associated with anger to remain constant in conceptualizing anger (and the emotions in general) throughout the ages because our experiential focus may change from culture to culture and through time.

More generally, what I would like to emphasize here is that universal embodiment associated with a target domain may consist of several distinct components or aspects. The conceptual metaphors that emerge may be based on one component, or aspect, at a certain point in history and on another at a different point. Which one comes to the fore depends on a variety of factors in the surrounding cultural context. In addition, the conceptual metaphors may be based on one component, or aspect, in one culture, while on another component, or aspect, in another culture. Moreover, even if there is a universal physiological component, the conceptualization of anger or other emotion concepts in a given language/culture may be based on related metaphors or metonymies only marginally. One such language is Tsou (an Austronesian language spoken in parts of Taiwan), where the emotions are primarily expressed linguistically through an elaborate prefix system attached to emotion *verbs* (not nouns). But as Shuanfan Huang (2002), the linguist who studied the language, tells us even in this language there exists the conceptual metaphor ANGER IS EXCESS AIR or FIRE IN A CONTAINER.

As a matter of fact, it also seems possible that universal physical or biological embodiment is entirely ignored in conceptualization. For example, we know of at least one culture where the angry

person is not, or is only to an insignificant degree, viewed as a pressurized container. Cathrine Lutz (1988) tells us that on Ifaluk, a Micronesian atoll, the folk conception of *song*, the counterpart of English anger, can be characterized in the following way:

1. There is a rule or value violation.
2. It is pointed out by someone.
3. This person simultaneously condemns the act.
4. The perpetrator reacts in fear to that anger.
5. The perpetrator amends his or her ways.

This model of *song* does not emerge from the mapping that characterizes the ANGRY PERSON IS A PRESSURIZED CONTAINER metaphor. The model emphasizes the *prosocial, moral, ideological* aspects of anger – as opposed to the antisocial, individualistic, and physical aspects that the PRESSURIZED CONTAINER metaphor emphasizes in Western cultures (Lutz, 1988). That is, although the Ifaluk physiology of anger may be very similar to the English and Chinese, this does not necessarily lead them to conceptualize *song* as pressure in a container. For the Ifaluk, anger is a much more social business, as their language, thinking, and behavior reveal. That is, *song* is an abstract concept motivated by the particular social–cultural practice of the Ifaluk, not by their bodily experience.

10.7 The HOT FLUID Metaphor in Relation to Embodiment, Culture, and Cognition

We have seen how embodiment, culture, and cognitive processes play a role in the conceptualization of anger. All three systems work jointly in the creation of particular metaphors and thus the metaphors may display a high degree of overall coherence. Let us take the ANGER IS A HOT FLUID IN A CONTAINER metaphor in English. First, as noted above, this metaphor is coherent with the bodily experience of anger. Second, it is also coherent with a particular system of social–cultural experience. Its coherence derives from the fact that this metaphorical conceptualization of anger is a social–cultural product deriving from the humoral view of emotions in Medieval Europe, and even earlier in Greek antiquity. Finally, it is coherent with a particular cognitive system – one that has a preferential experiential focus on the components of both heat and pressure, rather than just heat or just pressure. An example of the choice of heat only in the metaphorical conceptualization of anger would be a language that has heat-related expressions (like *hot-head*) but no HOT FLUID IN A CONTAINER metaphors, whereas an example of pressure only would be a language where pressure far outweighs heat in the conceptualization of anger. A language that comes fairly close to this latter situation is Chinese, as we saw above. The general point is that this metaphor is at the intersection of the three coherently interacting systems that were identified as playing a key role in metaphor variation in anger.

However, the picture is not as neat as we would like it to be. Take the ANGER IS A HOT FLUID IN concept of anger again. Much of the motivation for metaphorically conceptualizing anger as a HOT FLUID IN A CONTAINER comes from the physiological response of increase in body heat that people experience when they are in a state of intense anger. However, we can also talk about *cold anger* to refer to a particular kind of anger, say, when the angry person is meditating, in a self-controlled way, a retribution that far outweighs the offense. This kind of conceptualization of anger should not exist because it goes against the embodiment of anger that involves body heat. But it does exist and needs to be accounted for. In this case, I believe that the explanation is fairly straightforward. The notion of cold anger is based on conceptualizing a part of anger (retribution) as a rational act on the part of the angry person. It is this rational, as opposed to an emotional, decision that is conceptualized as

being “cold.” It is the RATIONAL IS COLD (as opposed to the EMOTIONAL IS HOT) metaphor that applies to a part of the cultural model of anger. In other words, I would claim that in this case the conflict in metaphorical conceptualization is more apparent than real.

10.8 Conclusions

In this chapter, I provided some evidence for the embodied nature of the concept of anger and some of its metaphors from work in cognitive psychology. This was necessary because my purpose was to show that some anger metaphors and some aspects of anger are near-universal. Indeed, many unrelated languages and cultures do seem to share the generic-level metaphor THE ANGRY PERSON IS A PRESSURIZED CONTAINER.

This metaphor plays a key role in structuring the concept in English and its counterparts in other languages and cultures. It underlies the widespread conception that anger is a force that makes the angry person perform aggressive or violent actions. The actual physiology of anger provides much support for this conceptualization.

But despite the universality of the physiology, the widespread nature of its metonymic and metaphoric conceptualization, and the near-universality of the generic-level cognitive model of anger, we have seen that there is a considerable amount of variation in the counterparts of anger both cross-culturally and intraculturally.

To account for some of this variation, a new, more nuanced view of embodiment was introduced, where the major idea is that the embodiment of anger consists of multiple components, and cultures may choose which of these components they focus on. This is what I called “experiential focus.” This idea helps in part explain why, despite physiology which may be universal, different cultures can have widely different understandings of their anger-like experiences.

References

- Barcelona, A. (2001). On the systematic contrastive analysis of conceptual metaphors: Case studies and proposed methodology. In M. Pütz, ed., *Applied cognitive linguistics II: Language pedagogy* (117–146). Berlin: Mouton.
- Bokor, Z. 1997. *Body-based constructionism in the conceptualization of anger* (C.L.E.A.R. Series, No. 17). Budapest: Department of English, Hamburg University and the Department of American Studies, ELTE.
- Csordas, T. (1994). *Embodiment and experience*. Cambridge and New York: Cambridge University Press.
- Ekman, P., Levenson, R. W., & Friesen, W. V. (1983). Autonomic nervous system activity distinguishes among emotions. *Science*, 221, 1208–1210.
- Fauconnier, G. & Turner, M. (2002). *The way we think. Conceptual blending and the mind's hidden complexities*. New York: Basic Books.
- Geeraerts, D. & Grondelaers, S. (1995). Looking back at anger: Cultural traditions and metaphorical patterns. In J. Taylor & R. MacLaury (Eds.), *Language and the cognitive construal of the world* (pp. 153–179). Berlin: Gruyter.
- Gevaert, C. (2001). Anger in old and middle english: A ‘hot’ topic? *Belgian Essays on Language and Literature*, 89–101.
- Gevaert, C. (2005). The ANGER IS HEAT question: Detecting cultural influence on the conceptualization of anger through diachronic corpus analysis. In N. Delbecque, J. van der Auwera, & D. Geeraerts (Eds.), *Perspectives on variation: Sociolinguistic, historical, comparative* (pp. 195–208). Berlin and New York: Mouton de Gruyter.
- Gibbs, R. W. (1992). Why idioms mean what they do? *Journal of Memory and Language*, 31, 485–506.
- Gibbs, R. W. (1994). *The poetics of mind. Figurative thought, language, and understanding*. Cambridge and New York: Cambridge University Press.
- Gibbs, R. W. (1999). Taking metaphor out of our heads and putting it into the cultural world. In R. Gibbs & G. Steen (Eds.), *Metaphor in cognitive linguistics* (pp. 146–166). Amsterdam: John Benjamins.
- Gibbs, R. W. (2006). *Embodiment and cognitive science*. New York and Cambridge: Cambridge University Press.

- Gibbs, R. W., Bogdonovich, J., Sykes, J., & Barr, D. (1997). Metaphor in idiom comprehension. *Journal of Memory and Language*, 37, 141–154.
- Gibbs, R. W. & O'Brian, J. (1990). Idioms and mental imagery: The metaphorical motivation for idiomatic meaning. *Cognition*, 36, 35–68.
- Grady, J. (1997a, b). THEORIES ARE BUILDING revisited. *Cognitive Linguistics*, 8, 267–290.
- Grady, J. 1997b. *Foundations of meaning: Primary metaphors and primary scenes*. Ph.D. dissertation, Department of Linguistics, University of California, Berkeley.
- Huang, S. (2002). Tsou is different: A cognitive perspective on language, emotion, and body. *Cognitive Linguistics*, 13(2), 167–186.
- King, B. 1989. *The conceptual structure of emotional experience in Chinese*. Ph.D. dissertation, Ohio State University.
- Kövecses, Z. (1986). *Metaphors of anger, pride, and love: A lexical approach to the study of concepts*. Amsterdam: Benjamins.
- Kövecses, Z. (1988). *The language of love: The Semantics of passion in conversational English*. Lewisburg, PA: Bucknell University Press.
- Kövecses, Z. (1990). *Emotion concepts*. Berlin and New York: Springer-Verlag.
- Kövecses, Z. (2000a). *Metaphor and emotion*. New York and Cambridge: Cambridge University Press.
- Kövecses, Z. (2000b). The scope of metaphor. In A. Barcelona (Ed.), *Metaphor and metonymy at the crossroads* (pp. 79–92). Berlin: Mouton.
- Kövecses, Z. (2002). *Metaphor: A practical Introduction*. New York and Oxford: Oxford University Press.
- Kövecses, Z. (2005). *Metaphor in culture*. New York and Cambridge: Cambridge University Press.
- Kövecses, Z. & Radden, G. (1998). Metonymy: Developing a cognitive linguistic view. *Cognitive Linguistics*, 9(7), 37–77.
- Lakoff, G. (1987). *Women, fire, and dangerous things. What categories reveal about the mind*. Chicago: The University of Chicago Press.
- Lakoff, G. & Johnson, M. (1980). *Metaphors we live by*. Chicago: The University of Chicago Press.
- Lakoff, G. & Kövecses, Z. (1987). The cognitive model of anger inherent in American English. In D. Holland & N. Quinn (Eds.), *Cultural models in language and thought* (pp. 195–221). Cambridge: Cambridge University Press.
- Lakoff, G. & Turner, M. (1989). *More than cool reason. A field guide to poetic metaphor*. Chicago: The University of Chicago Press.
- Levenson, R. W., Carstensen, L. L., Friesen, W. V., & Ekman, P. (1991). Emotion, physiology, and expression in old age. *Psychology and Aging*, 6, 28–35.
- Levenson, R. W., Ekman, P., & Friesen, W. V. (1990). Voluntary facial action generates emotion-specific autonomic nervous system activity. *Psychophysiology*, 27, 363–384.
- Levenson, R. W., Ekman, P., Heider, K., & Friesen, W. V. (1992). Emotion and autonomic nervous system activity in the Minangkabau of West Sumatra. *Journal of Personality and Social Psychology*, 62, 972–988.
- Lutz, C. (1988). *Unnatural emotions: Everyday sentiments on a micronesia atoll and their challenge to western theory*. Chicago: The University of Chicago Press.
- Maalej, Z. (2003). *The contemporary cognitive theory of metaphor: Arabic data*. Unpublished manuscript, University of Manouba, Tunisia.
- Matsuki, K. (1995). Metaphors of anger in Japanese. In J. R. Taylor & R. MacLaury (Eds.), *Language and the cognitive construal of the world* (pp. 137–151). Berlin: Mouton.
- Micholajczuk, A. (1998). The metonymic and metaphoric conceptualization of anger in Polish. In A. Athanasiadou & E. Tabakowska (Eds.), *Speaking of emotions: Conceptualization and expression* (pp. 153–191). Berlin: Mouton.
- Munro, P. 1991. *anger is heat: Some data for a cross-linguistic survey*. Manuscript, Department of Linguistics, UCLA.
- Palmer, G., Bennett, H., & Stacey, L. (1999). Bursting with grief, erupting with shame. A conceptual and grammatical analysis of emotion-tropes in Tagalog. In G. Palmer & D. Occhi (Eds.), *Languages of sentiment. Cultural constructions of emotional substrates* (pp. 171–200). Amsterdam: John Benjamins.
- Rosaldo, M. Z. (1980). *Knowledge and passion. Ilongot notions of self and social life*. Cambridge: Cambridge University Press.
- Soriano, C. (2003). *Conceptual metaphors and metonymies of anger in Spanish and English*. Paper presented at the 8th International Cognitive Linguistics Conference (ICLC). Spain: Logroño.
- Stearns, P. (1994). *American cool*. New York: New York University Press.
- Taylor, J. & Mbense, T. (1998). Red dogs and rotten mealies: How Zulus talk about anger. In A. Athanasiadou & E. Tabakowska (Eds.), *Speaking of emotions: Conceptualization and expression* (pp. 191–226). Berlin: Mouton.
- Yu, N. (1995). Metaphorical expression of anger and happiness in English and Chinese. *Metaphor and Symbolic Activity*, 10, 223–245.
- Yu, N. (1998). *The contemporary theory of metaphor: A perspective from Chinese*. Amsterdam: John Benjamins.

Part IV
Anger in Child Development

Chapter 11

The Development of Anger

Michael Lewis

Abstract This chapter describes the development of anger. To do so I need to distinguish between anger and other emotions and behaviors that are often confused with it. Having done so, I will argue that anger is an approach emotion. Unlike sadness or fear and more like happiness, its function is to move the infant into active engagement in its world in order to overcome obstacles to desired goals. Anger, as an approach action pattern, exists early and can be seen within the first few months of life.

Anger must be distinguished from aggression on the one hand and from rage on the other. This chapter differentiates anger from rage. Behaviorally, rage is more intense, diffuse, and prolonged. I argue that, ontogenetically, because rage involves the self-system, it does not emerge until after the first year and a half of life (Lewis, 1993). More important for my considerations, anger is the consequence of the blockage of a goal-directed action, while rage is the consequence of shame and is, therefore, a failure in the child's ability to maintain its self-esteem (Lewis, 1995). As I have argued elsewhere, shame cannot emerge until after the development of consciousness. Rage, therefore, cannot occur until shame arises, sometime after the second year of life (Lewis, 1992).

11.1 The Definition of Anger

To make sense of the concept of anger, it is necessary to appreciate that there are different kinds of anger – even though the various types are often lumped in a single category. Thus anger, aggression, rage, and willfulness have often been used interchangeably. Anger and willfulness have been taken up under the topic of narcissism. Much has been written about narcissism that may be relevant here (see Andrew Morrison's *Essential Papers on Narcissism*, 1986). Freud viewed narcissism in two ways: primary narcissism, which involves the initial libido investment of energy to the as yet undifferentiated ego, and secondary narcissism, which is withdrawal of psychic energy from objects back to the ego ([1914]1957). For Freud, primary narcissism is a normal action, a position similar to Heinz Kohut's (1972). Kohut argued that narcissism is not pathological but leads at the beginning of life to object love, that is, love for another. Kohut believed that narcissism, in its more mature form, led to other skills such as creativity, empathy, and humor. From another perspective,

M. Lewis (✉)
University of Medicine and Dentistry of New Jersey, Newark, NJ, USA
e-mail: lewis@umdnj.edu

and the perspective that I take here, we can think of narcissism, at least the nonpathological type, as a will to power (Nietzsche, [1904]1964; Rank, 1945), assertiveness (White, 1959), or even anger and intention (Lewis, 1990).

The difficulty in having at least two definitions of narcissism applies equally well to the term *anger*. We need to distinguish the two emotions of anger, anger and rage¹ as well as the term, “aggression.” I do not discuss aggression for it is usually seen as inappropriate and it is outside the scope of this chapter. Rather, I will first consider anger and rely on Darwin and others for its definition. Darwin considered anger as an emotion that “habitually leads to action” (1872, 78). “Anger and joy are, from the first, exciting emotions, and they naturally lead, more especially the former, to energetic movements which react on the heart and thus again on the brain” (p. 79). For Darwin, the most important feature of anger was its action orientation, the attempt of the organism to overcome an obstacle. This feature of anger, which includes facial expression, remains central to Darwin’s idea of the function of anger “The excited brain gives strength to the muscles, and at the same time, energy to the will” (p. 239). For Darwin, then, the function of anger is clear; it is action oriented and facially expressive. Izard (1977) picked up on this feature when he said that “Anger often results from physical or psychological restraint or from *interference* with goal-orientated activity . . . Readily mobilized energy tenses the muscles and provides a feeling of power, a sense of courage or confidence . . . The emotion of anger should be distinguished from actions of aggression” (p.87).

A central thesis of this chapter is that anger is first seen in relation to action aimed at overcoming barriers to goal-directed behavior. Anger is a natural and normal occurrence in all organisms, including infants, in their daily attempts to overcome barriers to desired goals. Some have likened it to will (Nietzsche, [1904]1964), some to efficacy (White, 1959), and others to power (Rank, 1945). Notice that this definition of anger is associated with restraint or interference with goal-orientated behavior and involved the muscles (or action) of the organism to overcome the blockage of the goal. Neither Darwin nor Izard distinguish between anger and rage, indeed they believe they differ only by degree. Here I will part company with them, because, for me anger, at least in the very young, is not a lesser form of rage. Anger is a restricted, focused response; rage is more intense, less focused, and longer lasting. Anger has a specific object, while rage tends to be diffused, both in terms of its occurrence and its object. Anger appears bounded, that is, there is a way to resolve it, whereas rage itself may be unbounded. Rage requires such elicitors as “personal insult . . . being taken advantage of, and being compelled to do something against one’s wishes” (Izard, 1977). When we think of an enraged person, we think of something having to do with serious intense psychological wounding or injury to the person’s feelings. Such an analysis almost immediately leads us to consider rage as a response to shame (Lewis, 1992). These elicitors cannot be present in the very young since they assume a level of objective self-awareness or consciousness that is not present until later, somewhere around 2 years of age.

The distinction I have made between anger and rage comes from a developmental perspective; it is quite clear that anger, as we measure it in the 8-week-old infant, cannot either in form or function be the same response that we see in older children or adults. Without careful analysis, we are likely to commit a serious error when we do not differentiate these terms. The existence of anger in the very young infants indicates that this is an innate action pattern whose chief function is its organized, motivating power to help organisms overcome barriers to desired goals. It is only later that this

¹ The term aggression is often used, but aggression may or may not be accompanied by anger or rage. Aggression makes reference to action toward another or others, whereas anger and rage speak to emotions as located within the individual.

emotion becomes elaborated to become anger in the adult form; that is anger that is still useful in obtaining desired goals but no longer an automatic and organized response to overcome a specific blockage to a goal. Finally, episodes of intense distress in early toddlerhood may be a precursor of the rage that humans feel when they are shamed. By making these distinctions and by careful articulation through differential language the difference between anger as an instrumental action, anger as an antisocial action, and anger as the consequence of shame we may go a long way in understanding not only the developmental sequence but also the routes of psychopathology.

My definition of anger, therefore, makes reference to a basic action pattern whose evolutionary adaptive function is to motivate the organism's action around overcoming obstacles to desired goals. Notice that in this definition there is no implication of the negative features that we usually attribute to this emotion. The form of anger that I address is the type most similar to the concept of will, efficacy, or even primary narcissism as already discussed. It is simply part of the very young organism's innate function to overcome obstacles. This action may take the form of behavior toward others or toward objects, but its primary function is its efficacy. It is, to use Nietzsche's phrase, the will to power, the will to action (1964). Until recently, angry expressions were thought to emerge in infants between 4 and 6 months of age. Note that the emergence of expressions of anger seems to coincide with the emergence of the child's mental capacity to learn the relationship between cause and effect or between action and outcome (Piaget, 1952). That anger expressions emerge at the same time as the children's capacity to learn how to affect their environment reinforces the idea of anger as a part of the action pattern designed to overcome a blocked goal. However, the blockage of a goal can occur, as we will see, anytime a means–end or action–outcome pairing occurs. In the natural world this may be around 4/6 months but under laboratory conditions this may be much earlier (Lewis, Alessandri, & Sullivan, 1990).

The examination of anger in young children historically has been indexed by increased instrumental responding. For example, John Watson (1925), M. Sherman, and I. Sherman (1925), and Sherman, Sherman, and Charles Flory (1936) reported an increased action elicited by arm and leg restraint. Infants younger than 6 months also respond to restraint by increased tension, breath holding, and by increased movement of the arms and hands. Sears and Sears (1940) and Marquis (1943), who examined frustration during interrupted feeding periods, also reported that blocking feeding resulted in immediate reaction characterized by increased defensive activity. This approach to the study of anger and its regulation continues using the contemporary "arm restraint" paradigm (e.g., Potegal, Robison, Anderson, Jordan, & Shapiro, 2007.)

These early reports collectively suggest that inducing frustration by restraining an infant's movement or by interrupting feeding behavior elicits increased motor activity and the occurrence of negative vocalizations. One of the limitations of this earlier research, however, was that facial expressions were not assessed. The limitation was due in part to the lack of discriminative measures of negative emotions and to the prevailing view that emotional behavior was relatively undifferentiated early in life (Bridges, 1932). Technological developments and refinement of facial expression coding systems have allowed for the assessment of infant facial expressions, although difficulties remain (see Camras, 2004).

More recently, anger expressions in response to a frustrating event have been examined in 4- to 6-month olds (Stenberg, 1982; Stenberg, Campos, & Emde, 1983). In 4-month-old infants, they occurred when arm movements are restrained and in 7-month olds when a teething biscuit was withdrawn from the baby's grasp. In addition, Stenberg et al. (1983) report that infants expressed more anger when mothers, rather than strangers, removed the biscuit and that repetition of the task increased the amount of anger expressed by the infant. These studies demonstrate that eliciting situations that block instrumental actions, such as sucking on or eating a biscuit, even in young infants, reliably elicit anger expressions.

The onset of anger between 4 and 6 months of age has been theoretically linked to the development of means–end ability. Means–end ability refers to the infant’s understanding of the relationship between his or her own activity and a desired object or goal. This understanding is thought to develop over the first 2 years of life, from simple body-centered actions to more flexible and insightful goal-directed behavior toward objects or goals (Piaget, 1952). Anger is the emotional response typically associated with the blockage of activity toward an expected goal (Plutchik, 1980; Stein & Jewett, 1986) and should be related to the young infants’ emerging means–end knowledge. Thus, for example, Izard, Hembree, and Huebner (1987) observed a response of general distress to a pain event in infants up to 7 months of age. Thereafter, anger, rather than a pain response, was predominant. Stenberg et al. (1983) likewise reported anger to frustration at 4–7 months. For anger to occur, it may be necessary that the infant be able to associate the blockage of the goal with the source of the failure to obtain that goal. It makes little adaptive sense to express emotional responses related to overcoming a blockage to a goal without being able to recognize a means related to that goal.

Thus, certainly by 4 months of age and after, the emotional response of anger, including facial expression and motor action, is likely to be expressed. However, it may be any time an organism can learn a relationship between an action and an outcome, when that action is blocked, an angry response may be expected. An angry response should be related not only to the general ability of children to establish means–end relationships – something that Piaget (1952) argued occurs after 4 months of age – but should occur *whenever the infant has learned a response to a desired goal that is interrupted*. Thus if a learned response to a goal occurs even before 4 months, when interrupted, it should result in anger. The work of Lewis and associates indicates this to be the case.

11.2 Studies of the Response to Goal Blockage

The focus of our research has been on understanding individual differences in the goal blockage response itself. We have shown that individual differences in anger and sadness occur early in life and are stable from 2 to 8 months (Lewis, Sullivan, Ramsay, and Alessandri, 1992; Sullivan, Lewis, & Alessandri, 1992). Infants in this age range are able to learn to pull a ribbon to activate an audiovisual event (Alessandri, Sullivan, & Lewis, 1990). To assess individual differences, they are first trained in this contingency, then experience a brief period when their access to the event is blocked (extinction). Anger expressions occur when access to the contingent goal is blocked but some infants show sadness as well. Infants who show anger in response to blockage resume pulling and show interest when access to the contingency is restored, whereas those who show sadness do not resume pulling and show low enjoyment when access is restored (Lewis et al., 1992). We also have developed procedures to assess autonomic responses to goal blockage which include heart rate and heart rate variability as well as cortisol release. Using these methods, we find individual differences in expression, behavioral responses, and differential physiological patterns: Anger expressions and increased instrumental behavior are related to physiological patterns which energize the individual. This includes increased cardiac output but not cortisol activity (Chapter 7 by Stemmler, this book). In contrast, sadness and decreased action toward the goal are supported by the physiological patterns which serve passive withdrawal or inhibition; that is greater cortisol release, but not cardiac activation. We have also found increased heart rate and decreased heart rate variability in response to goal blockage that accompanies anger and increased attempts to regain the goal. Lewis & Ramsay (2005) and Lewis, Ramsay, & Sullivan (2006) provide evidence of differential cardiac and cortisol responses related to anger and sadness, thus supporting the idea of different action patterns of approach and withdrawal.

We have also varied the blockage procedure, using social stimuli as in the mother–children “en face” procedure as well as the string pulling blockage to assess the differential relation of anger and sadness to the cortisol response (Lewis & Ramsay, 2005). The blocked goal is the mother’s sudden dropping of her head, which terminates the social interaction. We find that regardless of whether it is a social or an instrumental condition, sadness was related, but anger was unrelated, to greater cortisol release. There is a considerable body of work on individual differences in adrenocortical response in relation to goal blockage. Both theory and research suggest that a cortisol response to goal blockage is likely to occur primarily when there is an inability to overcome the obstacle or to be able to regain the goal. That is when there is a perceived lack of control (Levine, Coe, & Weiner, 1989). Lack of control also typically results in attempts to withdraw or inhibit behavior as observed in our own work and that of others (DeCapser & Carstens, 1981; Lewis, Hitchcock, & Sullivan, 2004; Seligman, 1975b; Sullivan & Lewis, 2003). If anger to a blocked goal reflects some perceived ability to regain the goal, it should not be associated with increases in cortisol. Conversely, if sadness to a blocked goal reflects some perceived lack of ability to regain the goal, it is likely to be associated with increases in cortisol. Our cortisol and cardiac data therefore converge with theory in suggesting that different physiological systems are associated with individual differences in anger and sadness.

11.3 Emotions as Approach and Withdrawal

There is a larger theoretical framework for understanding the experimental results described above, namely, that anger and sadness represent approach and withdrawal motivations, respectively. Approach and withdrawal have long been viewed as basic motivational tendencies with individual differences having important consequences for children’s subsequent development (Darwin, 1965; Schneirla, 1959). The recent literature on central nervous system organization of emotion also supports the view that these motivational action tendencies underlie emotion. Approach and withdrawal action tendencies have been related to goal pursuit and goal blockage and are thought to link emotional expression, physiological patterns, and evaluative behavior (Carver, Sutton, & Scheier, 2000; Lewis & Ramsay, 2005). Approach and withdrawal emotional systems evolved from primitive neural circuits and are differentially related to specific emotions observed in humans and mammals. Panksepp (1998) has suggested that sadness promotes inhibition, passive withdrawal, or giving up in contrast to the exploratory or “seeking system” which motivates goal approach and active pursuit. Studies examining approach and withdrawal in infancy generally support the view that approach and withdrawal are linked to different expression patterns and are functionally different systems (Davidson, 1998; Fox & Davidson, 1988; Putnam & Stifter, 2005). Carver (2004) further distinguished between withdrawal emotion in approach contexts (sadness) and avoidance in threat contexts (fear). Although both can be characterized as inhibitory affects, he proposes that they may be separate processes. This view is supported by studies that distinguish between behavioral inhibition and social withdrawal/anxiety (Ballespi, Jane, Riba, & Domenech-Liaberia, 2002; Kerr, Tremblay, Pagani, & Vitaro, 1997). Others, however, suggest that they are part of a single biphasic avoidance/withdrawal system in which sadness emerges only after prolonged exposure to stress (Hennessy, Deak, & Schiml-Webb, 2001). This view is supported by work showing that sadness and depression-like responses emerge after prolonged exposure to stressors which cannot be controlled (Seligman, 1975a). The literature has been consistent in suggesting that children can be to some degree identified from infancy who are either high in approach tendency (anger) versus those who are high in inhibition (fear) or withdrawal (sadness). In fact, behavioral inhibition can be understood as having a low threshold for avoidance and may be related to both fear and sadness (Buss,

Davidson, Kalin, & Goldsmith, 2004; Buss & Kiel, 2004). The literature has not typically distinguished between sadness and fear processes prior to 2 years of age. This may be due to a widely held view that negative emotion is not differentiated before 6–7 months. However, our work consistently has demonstrated that individual differences in anger and sadness occur in response to a blocked goal (Alessandri, Sullivan, Imaizumi, & Lewis, 1993; Lewis et al., 1990, 1992; Sullivan & Lewis, 2003; Sullivan et al., 1992; Lewis & Ramsay, 2005).

Infant expressions are only one aspect of emotional systems. Emotional systems involve the coordination of expressivity, physiology, behavior, and cognition which occurs in particular contexts (Lewis, Sullivan, & Michalson, 1984). I have suggested that infancy is an ideal time to study the origins of individual differences in the organization of these systems, as most measures are noninvasive and infants' expressions of emotion are not yet influenced by display rules and before learned coping strategies come into play (Lewis et al., 1990, 1992; Sullivan & Lewis, 2003).

11.4 Anger and the Approach System

Darwin classed anger with joy as an “exciting” or approach emotion as opposed to “depressing” or withdrawal emotion. Panksepp (1998) also views anger as closely linked to the approach/exploratory system and regulator of responses to goal blockage in mammals and primates. A number of studies have examined EEG activation to various forms of anger induction (e.g., Waldstein et al., 2000, Chapter 5 by Harmon-Jones et al., this book.) In general, work with adults and children as young as 6 months has demonstrated left-prefrontal cortical activity is associated with approach motivation while right-prefrontal cortical activity is associated with withdrawal motivation (Coan, Allen, & Harmon-Jones, 2001; Harmon-Jones, 2003). Recent work on anger reveals that both trait and state anger and positive, but not negative, assertiveness are related to increased left frontal CNS activation (Harmon-Jones, 2004; Hewig, Hagemann, Seifert, Naumann, & Bartussek, 2004; Wacker, Heldmann, & Stemmler, 2003) and lower cortisol and cardiovascular reactivity under heightened performance pressure (Lerner, Gonzalez, Dahl, Hariri, & Taylor, 2005). Our own work suggests the toddler trait of determination is related to greater approach at 5 months and support the view that anger shares the CNS pattern associated with approach emotions and is distinct from withdrawal and inhibition (Buss & Goldsmith, 1998; Buss, Schumacher, Dolski, Kalin, & Davidson, 2003). Because direction of adaptive action (approach) and emotional valence (negative) are confounded in anger (Harmon-Jones, 2004), studies using contexts in which anger is an approach action tendency are particularly relevant. It is these that we have studied.

Anger has not typically been considered as an approach tendency in the infant literature despite general acceptance of opponent process models of emotion in human and animal work (Solomon, 1977; Suomi, Mineka, & Harlow, 1983). Negative facial and vocal expressions and their regulation in infancy have been studied primarily from the perspective of aversive contexts. Stimuli used, with a few exceptions (Braungart-Reiker & Stifter, 1996; Calkins & Johnson, 1998), have been novel, and potentially threatening or intrusive, so as to induce the inhibition/withdrawal emotion systems (Fox, Henderson, Rubin, Calkins, & Schmidt, 2001; Putnam & Stifter, 2005). Such paradigms do not accommodate the view of anger as an approach action tendency, or consider that it may be a contextually appropriate response to goal blockage. Only a few have recognized that “distress” is too global a construct in infant emotion regulation (Buss & Goldsmith, 1998). The infant emotion field has found few contexts which elicit predominantly anger in young infants, the contingency blockage procedure being the notable exception. The arm restraint procedure (Camras, Oster, Campos, Miyake, & Bradshaw, 1992; Stenberg & Campos, 1990), another putative anger elicitor, is not clearly a goal

blockage context, perhaps accounting for inconclusive data observed at 4 months. In contrast, our work finds stable individual differences in anger to goal blockage between 2 and 8 months (Sullivan et al., 1992). Thus, our findings on goal blockage offer a perspective on infant emotion which tap into the approach aspect of the system.

11.5 Sadness and the Withdrawal System

In contrast to anger, sadness was viewed by Darwin as an emotion of withdrawal, in particular when the “sufferer is conscious that nothing can be done”. The relation of lack of control to depression and helplessness (withdrawal) has been recognized in infants, children, and adults (DeCapser & Carstens, 1981; Jennings, 2004; Rothbaum, Wolfer, & Visintainer, 1980; Seligman, 1991). Sad expressions are significantly likely during maternal bids for help in 2-year olds (Buss & Kiel, 2004). Loss of control, uncertainty, and passive responding in the face of difficulty all have been linked to long-lasting alterations in cortisol response (Henry, 1992). The withdrawal emotional system has been strongly linked to differences in cortisol release and to greater anxiety and depression risk. Individual differences in the withdrawal system may be an important way to identify risk of future psychopathology (Bauer, Quas, & Boyce, 2002; Gotlib, Joormann, Minor, & Cooney, 2006). In infants, fear and sadness behaviors have been linked both to cortisol release and EEG asymmetry (Buss et al., 2003). Our findings show that sadness and cortisol release are related and that sadness is more likely when control of an event has been lost (Sullivan & Lewis, 2003). Our findings support the view that both sad expressions and cortisol release are aspects of the withdrawal system.

To summarize, approach and withdrawal systems exhibit distinctive action tendencies, facial expressions, and physiological organization in which anger and sadness play a major part. Individual differences in approach and withdrawal emerge in infants by 2–5 months.

11.5.1 Outcomes Related to Approach and Withdrawal

Individual differences in anger/approach and sad/withdrawal systems may have implications for normal and dysfunctional development. We have observed the relation between individual differences in anger/approach and sadness/withdrawal at 5 months and behavioral outcomes at ages 2 and 3 years. Greater infant anger to goal blockage is related to measures of persistence and determination in toddlers and *fewer* behavioral difficulties at 3 years (Sullivan, 2007). Collectively, our results show that infants’ responses to blocked goals are a window to the study of individual differences in the anger/approach and sadness/withdrawal emotional systems. Whether individual differences in approach and withdrawal in infancy continue to predict later aspects of personality and resilience beyond early childhood is an important theoretical and practical question from the perspectives of behavior management and clinical outcomes.

Personality differences have long been hypothesized to have their origins in individual differences in emotionality (Lewis & Michalson, 1983). Differences in early approach and withdrawal emotional systems are manifested as later differences in persistence and other aspects of behavioral style (Lewis & Ramsay, 2005). Mastery motivation, for example, has relevance for the study of approach and withdrawal emotional systems since the mastery motive has been defined as a desire to explore and achieve control over one’s environment (MacTurk & Morgan, 1995; Turner & Johnson, 2003). Persistence and individual differences in persistence are apparent by late infancy and serve as the primary index of early mastery motivation (MacTurk & Morgan, 1995; Messer, 1993). Children

as young as 3 years who persist in the face of challenge demonstrate a sense of control over the situation whereas children who withdraw from tasks do not feel they have control over outcomes and exhibit helplessness (Dweck, Chiu, & Hong, 1995; Lutkenhaus, 1984). Measures of persistence, as opposed to helplessness, are likely to be associated with approach motivation at least into middle childhood (Dichter-Blancher, Busch-Rossnagel, & Knauf-Jensen, 1997; Eisenberg et al., 2005). Thus differences in approach and withdrawal systems at 5 months should be related to subsequent behavioral styles of persistence and helplessness.

Our findings on the relation between 5-month anger/approach and toddler persistence and determination support the view of the relation between early differences in anger and sadness and later behavioral styles of persistence, low helplessness, and efficacy. For example, there is a large body of work suggesting that infant behavioral inhibition is related to greater risk of behavioral difficulties (Calkins & Fox, 1992; Kagan, 1994; Rubin, Hastings, Stewart, Henderson, & Chen, 1997). Differences in autonomic and adrenocortical responses related to poor mental health outcomes in childhood also appear to map onto approach and withdrawal emotion systems. Social withdrawal in boys is related to depression at age 15, whereas attentional control or approach is protective (Kerr et al., 1997).

Does the early organization of approach and withdrawal systems related to later health? Type-A behavior, a behavioral risk factor in hypertension has been studied in children as young as 4 years, and is correlated positively with reactivity, achievement need, hostility, and anxiety (Brown & Tanner, 1988; Matthews, Woodall, Engebretson, & McCann, 1992; Oginska-Bulik & Juczynski, 1998; Suarez, Williams, Kuhn, Zimmerman, & Schanberg, 1991). At the same time, Type-A children also are reportedly more self-confident, vigorous, demanding, and impulsive (Oginska-Bulik & Juczynski, 1998). On the whole, this mixture of traits suggests greater sympathetic vs. parasympathetic cardiac activation and greater approach relative to withdrawal in children with the Type-A pattern. Type-A children's systolic and diastolic blood pressures are also significantly correlated with adrenaline excretion between 3 and 6 years (Lundberg, Rasch, & Westermarck, 1990) and to greater rises in systolic blood pressure in response to challenge as well as later hypertension (Brown & Tanner, 1988; Sallis, Dimsdale, & Chipman, 1988, see Williams, this book for review of these issue in adults). Clearly more work needs to be done to relate approach/withdrawal styles with physical health.

11.6 Sources of Individual Differences in Approach and Withdrawal at 5 Months

11.6.1 Experiential Factors

Although individual differences in approach and withdrawal observed at 5 months might originate in innate physiological patterns present in the early postnatal period, responsive caregiver interactions may entrain individual differences in approach and withdrawal systems, or certain early physiological patterns in combination with responsive parenting may promote these differences. Several theoretical views and much empirical data on early maternal behavior show that sensitive and responsive behavior is likely to influence the organization of the approach and withdrawal systems. Caregiver behavior, usually maternal sensitivity and responsiveness, is associated with more optimal emotional outcomes including attachment security (Belsky, 1997; Fox, Kimmerly, & Schafer, 1991; Goldsmith & Alansky, 1987). Attachment theory suggests that caregiver sensitivity has a direct influence on approach and withdrawal responses to mother following a separation (Ramsay,

Sullivan, & Lewis, 2005), since sensitive caregiving is believed to foster infants' modulation of arousal (Ainsworth, Blehar, Waters, & Wall, 1978; Field, 1994; Sroufe, 1996; Tronick, 1989).

Maternal insensitivity on the other hand is generally found to be related to greater cortisol response and to greater withdrawal (Liu et al., 1997; Nachimas, Gunnar, Mangelsdorf, Parritz, & Buss, 1996; Spangler & Grossman, 1993; Spangler, Schieche, Lllg, Maier, & Ackerman, 1994). Lewis and Goldberg (1969) argued that early responsivity results in a generalized expectancy of a responsive environment. This theoretical view, like attachment, implies that sensitive, responsive caregiving will be related to relatively greater approach than withdrawal emotions (Lewis & Michalson, 1983; Malatesta & Haviland, 1982) and ultimately to a generalized sense of self-efficacy and the predictability of the world (Brinker & Lewis, 1982; Lewis & Goldberg, 1969; Watson, 1972). Animal work investigating maternal influences on the reactivity and regulation components of cortisol release also suggests the differential impact of maternal behavior on approach and withdrawal (Frances et al., 1996; Hennessy, O'Leary, Hawke, & Wilson, 2002; Liu et al., 1997). There is a need to consider early maternal responsiveness/sensitivity alone and in interaction with early infant physiological patterns when considering the antecedents of approach and withdrawal.

11.6.2 Physiological Influences

Direct assessment of physiological patterns using multiple measures provides a unique opportunity to examine their relation to approach and withdrawal systems with greater precision than with a single physiological measure or maternal ratings of temperament. Because we find evidence that 5-month approach and withdrawal are differentially related to concurrent physiological organization, it is reasonable to ask whether early physiological patterns influence the organized approach and withdrawal patterns observed at 5 months. For example, the relation between cortisol level and later sadness would provide important evidence linking early physiology to the emergence of organized withdrawal responses.

In the adrenocortical system as well as in other systems, it is possible to obtain basal as well as reactivity measures (Cacioppo, Klein, Berntson, & Hatfield, 1993; Fox & Calkins, 1993). Studies typically examine individual differences in cortisol in relation to negative emotionality. Some work focusing on maltreated children and children of depressed mothers suggests aberrant cortisol levels (Gunnar, Morison, Chisholm, & Schuder, 2001; Hart, Gunnar, & Cicchetti, 1995; Huot, Brennan, Stowe, Plotsky, & Walker, 2004). High basal cortisol is a discriminating measure of behavioral inhibition in normally developing children (Kagan, Reznick, & Snidman, 1987; Schmidt et al., 1997). Behavioral inhibition may be one aspect of the withdrawal system. Differences in approach and withdrawal patterns among preschoolers have also been related to differences in basal cortisol and resting heart rate variability patterns (Blair, Peters, & Granger, 2004). However, past work has not focused on basal cortisol in relation to specific emotions in normative populations.

Measures of heart rate and heart rate variability provide stable and reliable early measures of individual differences during the infant and early childhood period (Berston, Cacioppo, & Quigley, 1993; Calkins, 2009; Lewis et al., 2006; Moore & Calkins, 2004; Richards, 1995). Cardiac autonomic functioning is thought to provide an index of approach and withdrawal from objects or people and individual differences in heart rate and heart rate variability during non-stress periods show associations with appropriate approach responses (Porges, 1998, 2001; Porges, Doussard-Roosevelt, Portales, & Greenspan, 1996). The relative dominance of sympathetic and parasympathetic influences on heart rate is thought to be particularly important to the approach and withdrawal emotional systems (Berston et al., 1993; Sahar, Shalev, & Porges, 2001). Greater activation of the "vagal

brake" (i.e., reduced heart rate and increased heart rate variability) during a challenge predicts later outcomes including less shyness (i.e., social inhibition/withdrawal) and greater sociability (social approach) in kindergarten children (Doussard-Roosevelt, 2003). Differences in non-stress heart rate variability patterns as well as amount of vagal suppression differentiate kindergarteners with externalizing as opposed to mixed behavior problem profiles (Calkins, 2009; Calkins & Keane, 2004; Calkins, Graziano, & Keane, 2007). In newborns, individual differences in non-stress heart rate variability has been positively correlated with successful developmental outcomes (Hofheimer, Wood, Porges, Pearson, & Lawson, 1995). Calkins (Calkins, Smith, Gill, & Johnson, 1998) hypothesized that individual differences in non-stress heart rate variability more directly reflect biological factors, whereas heart rate variability response to challenge is more responsive to environmental influence. However, no studies to my knowledge have examined the relation of early heart rate variability patterns to later approach and withdrawal.

Poorer heart rate variability regulation, change in response to challenge over time, is related to both defensiveness and low behavioral activation, characteristics of the withdrawal system (Allen, 2005). Direct tests of the vagal regulation model in infants and children have as yet been few, but our work and that of others, suggests that reciprocal pairing of heart rate variability with heart rate is observed during periods of approach (e.g., contingency learning, social engagement), and when heightened attention to abrupt changes in environmental stimulation occur (Bazhenova, Plonskaia, & Porges, 2001; Lewis et al., 2004; Porges et al., 1996). In our studies, decreased heart rate variability and increased heart rate are positively related to anger and to increased instrumental behavior, results consistent with the approach model proposed here (see Lewis et al., 2004; Porges et al., 1996).

Most studies relating individual differences in early cardiac organization or cortisol to emotion involved reactivity to stressful contexts such as fear of strangers, solving arithmetic problems, or working on a difficult puzzle while receiving negative feedback (Allen, 2005; Blair et al., 2004; Doussard-Roosevelt, 2003; Sahar et al., 2001; van Bakel & Riksen-Walraven, 2004). The results suggest that dysregulation of either system predicts behavioral difficulty, but few studies integrate cardiac and cortisol release measures and few child studies include separate sympathetic and parasympathetic measures; see Buss et al. (2004) for an exception.

11.7 Summary

To study children's anger we must make sure that it is anger, not rage or aggression, which is being elicited since these concepts have been badly confused. Like Darwin, I believe that anger is an approach response to a blocked goal. In general, a child will react with anger when she/he knows of the response-goal connection and that it is blocked. If the child does not know the connection between a response and an outcome, then its blockage will not necessarily elicit anger. Piaget argued that a means-end ability must develop, but did not emerge until 4 months of age under normal conditions. Our use of a non-ecological paradigm of connecting arm pull response and picture presentation may facilitate the earlier development of a means-end connection. Once having established a means-end connection, its blockage should, in general, lead to anger, a finding that is suggested by our work. We have been able to show this effect as early as 2 months of age and the patterns seen at 2 months are the same as those shown at 8 months (Lewis et al., 1990).

These findings suggest that the capacity for anger develops soon after the first month of life. While different goal blockages occur as the infant develops, it appears as if the response of anger, an approach to overcome the blockage of the goal, is established early and does not undergo developmental transformations. However, like all emotional primitives its motivational properties can be

captured and used by the social environment as the basis of other action patterns. Like the primitive action pattern of disgust, originally evolved as a device to expel unpleasant tastes and smells, but later captured and used in moral disgust (the disgust at an idea), the action pattern of anger can be socialized to produce persistence, concentrated effort, and problem solving on the one hand or to produce rage and aggression to others.

References

- Ainsworth, M. D. S., Blehar, M. C., Waters, E., & Wall, S. (1978). *Patterns of attachment: A psychological study of the strange situation*. Hillsdale, NJ: Erlbaum.
- Alessandri, S. M., Sullivan, M. W., Imaizumi, S., & Lewis, M. (1993). Learning and emotional responsivity in cocaine-exposed infants. *Developmental Psychology*, 29(6), 989–997.
- Alessandri, S. M., Sullivan, M. W., & Lewis, M. (1990). Violation of expectancy and frustration in early infancy. *Developmental Psychology*, 26(5), 738–744.
- Allen, J. B. (2005). Cardiac vagal tone, defensiveness, and motivational style. *Biological Psychology*, 68(2), 147–162.
- Ballesi, S., Jane, M. C., Riba, M. D., & Domenech-Liaberia, E. (2002). Analysis of anxious and depressive symptoms of a sample of inhibited preschoolers. *Revista de Psiquiatria Infanto-Juvenil*, 19(4), 184–191.
- Bauer, A. M., Quas, J. A., & Boyce, W. T. (2002). Associations between physiological reactivity and children's behavior: Advantages of a multisystem approach. *Developmental and Behavioral Pediatrics*, 23(2), 102–113.
- Bazhenova, O., Plonskaia, O., & Porges, S. W. (2001). Vagal reactivity and affective adjustment in infants during interaction challenges. *Child Development*, 72, 1314–1326.
- Belsky, J. (1997). Theory, testing, effect-size evaluation, and differential susceptibility to rearing influence: The case of mothering and attachment. *Child Development*, 64, 598–600.
- Berston, G. G., Cacioppo, J. T., & Quigley, K. S. (1993). Respiratory sinus arrhythmia: Autonomic origins, physiological mechanisms and psychological implications. *Psychophysiology*, 30, 183–196.
- Blair, C., Peters, R., & Granger, D. (2004). Physiological and neuropsychological correlates of approach/withdrawal tendencies in preschool: Further examination of the behavioral inhibition system/behavioral activation system scales for young children. *Developmental Psychobiology*, 45(3), 113–124.
- Braungart-Reiker, J. M., & Stifter, C. (1996). Infant responses to frustrating situations. Continuity and change in reactivity and regulation. *Child Development*, 67, 1767–1779.
- Bridges, K. L. (1932). Emotional development in early infancy. *Child Development*, 3, 324–341.
- Brinker, R., & Lewis, M. (1982). Making the world work: A learning prosthesis for handicapped infants. *Exceptional Children*, 49, 163–170.
- Brown, M. S., & Tanner, C. (1988). Type A behavior and cardiovascular responsivity in preschoolers. *Nursing Research*, 37(3), 152–155.
- Buss, K. A., Davidson, R. J., Kalin, N. H., & Goldsmith, H. H. (2004). Context-specific freezing and associated physiological reactivity as a dysregulated fear response. *Developmental Psychology*, 40(4), 583–594.
- Buss, K. A., & Goldsmith, H. H. (1998). Fear and anger regulation in infancy: Effects on the temporal dynamics of affective expression. *Child Development*, 69, 359–374.
- Buss, K. A., & Kiel, E. J. (2004). Comparison of sadness, anger, and fear facial expressions when toddlers look at their mothers. *Child Development*, 75(6), 1761–1773.
- Buss, K. A., Schumacher, J. W., Dolski, I., Kalin, N. H., & Davidson, R. J. (2003). Right frontal brain activity, cortisol, and withdrawal behavior in 6-month-old infants. *Behavioral Neuroscience*, 117(1), 11–20.
- Cacioppo, J. T., Klein, D. J., Berntson, G. G., & Hatfield, E. (1993). The psychophysiology of emotion. In M. Lewis & J. M. Haviland (Eds.), *Handbook of emotion* (pp. 119–142). New York: Guilford Press.
- Calkins, S. D. (2009). Regulatory competence and early disruptive behavior problems: The role of physiological regulation. In S. Olson, & A. Sameroff (Eds.), *Biopsychosocial regulatory processes in the development of childhood behavioral problems* (pp. 86–115). NY, NY: Cambridge University Press.
- Calkins, S. D., & Fox, N. (1992). The relations among infant temperament, security of attachment, and behavioral inhibition at twenty-four months. *Child Development*, 63, 1456–1472.
- Calkins, S. D., Graziano, P. A., & Keane, S. P. (2007). Cardiac vagal regulation differentiates among children at risk for behavior problems. *Biological Psychology*, 74(2), 144–153.
- Calkins, S. D., & Johnson, M. C. (1998). Toddler regulation of distress to frustrating events: Temperamental and maternal correlates. *Infant Behavior and Development*, 21(3), 379–395.

- Calkins, S. D., & Keane, S. P. (2004). Cardiac vagal regulation across the preschool period: Stability, continuity, and implications for childhood adjustment. *Developmental Psychobiology*, 45, 101–112.
- Calkins, S. D., Smith, C. L., Gill, K. L., & Johnson, M. C. (1998). Maternal interactive style across contexts: Relations to emotional behavioral, and physiological regulation during toddlerhood. *Social Development*, 7(3), 350–369.
- Camras, L. (2004). An event-emotion or event-expression hypothesis? A comment on the commentaries on Bennett, Bendersky, and Lewis (2002). *Infancy*, 6(3), 431–433.
- Camras, L., Oster, H., Campos, J. J., Miyake, K., & Bradshaw, D. (1992). Japanese and American infants' responses to arm restraint. *Developmental Psychology*, 28(4), 578–583.
- Carver, C. S. (2004). Negative affects deriving from the behavioral approach system. *Emotion*, 4(1), 3–22.
- Carver, C. S., Sutton, S. K., & Scheier, M. F. (2000). Action, emotion, and personality: Emerging conceptual integration. *Personality & Social Psychology Bulletin*, 26(6), 741–751.
- Coan, J. A., Allen, J. J. B., & Harmon-Jones, E. (2001). Voluntary facial expression and hemispheric asymmetry over the frontal cortex. *Psychophysiology*, 38(6), 912–925.
- Darwin, C. (1872). *The expression of the emotions in man and animals*. Chicago: University of Illinois Press.
- Darwin, C. (1965). *The expression of emotion in man and animals*. Chicago: University of Chicago Press.
- Davidson, R. (1998). Affective style and affective disorders: Perspectives from neuroscience. *Cognition and Emotion*, 12, 307–330.
- DeCapser, A. J., & Carstens, A. A. (1981). Contingencies of stimulation: Effects of learning and motivation. *Infant Behavior and Development*, 4, 19–35.
- Dichter-Blancher, T. B., Busch-Rossnagel, N. A., & Knauf-Jensen, D. E. (1997). Mastery motivation: Appropriate tasks for toddlers. *Infant Behavior and Development*, 20, 545–548.
- Doussard-Roosevelt, J. A. (2003). Short-term stability of physiological measures in kindergarten children: Respiratory sinus arrhythmia, heart period, and cortisol. *Developmental Psychobiology*, 43(3), 231–242.
- Dweck, C., Chiu, C., & Hong, Y. (1995). Implicit theories: Implicit theories and their role in judgments and reactions: A world from two perspectives. *Psychological Inquiry*, 6, 267–285.
- Eisenberg, N., Qing, A., Spinrad, T. L., Valiente, C., Fabes, R. A., & Liew, J. (2005). Relations among positive parenting, children's effortful control and externalizing problems: A three wave longitudinal study. *Child Development*, 76(5), 1055–1071.
- Field, T. (1994). The effects of mother's physical and emotional unavailability on emotion regulation. In N. A. Fox (Ed.), *Monographs of the society for research on child development* (Vol. 59, Serial No. 240, pp. 208–227). Chicago, IL: The University of Chicago Press.
- Fox, N., & Calkins, S. D. (1993). Pathways to aggression and social withdrawal: Interactions among temperament, attachment, and regulation. In K. H. Rubin & J. Asendorf (Eds.), *Social withdrawal, shyness and inhibition in childhood* (pp. 81–100). Hillsdale, NJ: Lawrence Erlbaum.
- Fox, N., & Davidson, R. (1988). Patterns of brain electrical activity during facial signs of emotion in 10-month-old infants. *Developmental Psychology*, 24(2), 230–236.
- Fox, N., Henderson, H. A., Rubin, K. H., Calkins, S. D., & Schmidt, L. A. (2001). Continuity and discontinuity of behavioral inhibition and exuberance: Psychophysiological and behavioral influences across the first four years of life. *Child Development*, 72, 1–21.
- Fox, N., Kimmerly, N. L., & Schafer, W. D. (1991). Attachment to mother, attachment to father: A meta-analysis. *Child Development*, 62, 210–225.
- Frances, D., Diorio, J., LaPlante, P., Weaver, S., Secki, J. R., & Meaney, M. J. (1996). The role of early environmental events in regulating neuroendocrine development: Moms, pups, stress, and glucocorticoid receptors. *Annals of the New York Academy of Sciences*, 794, 136–152.
- Freud, S. ([1914]1957). *On narcissism: An Introduction* (Standard Ed., Vol. 14, pp. 67–102). London: Hogarth Press.
- Goldsmith, H. H., & Alansky, J. A. (1987). Maternal and infant temperamental predictors of attachment: A meta-analytic view. *Journal of Clinical and Consulting Psychology*, 55, 805–816.
- Gotlib, I. H., Joormann, J., Minor, K. L., & Cooney, R. E. (2006). Cognitive and biological functioning in children at risk for depression. In T. Canli (Ed.), *Biology of personality and individual differences*. New York: Guilford Press.
- Gunnar, M. R., Morison, S. J., Chisholm, K., & Schuder, M. (2001). Salivary cortisol levels in children adopted from Romanian orphanages. *Development and Psychopathology*, 13, 611–628.
- Harmon-Jones, E. (2003). Clarifying the emotive functions of asymmetrical frontal cortical activity. *Psychophysiology*, 40(6), 838–848.
- Harmon-Jones, E. (2004). Contributions from research on anger and cognitive dissonance to understanding the motivational functions of asymmetrical frontal brain activity. *Biological Psychology*, 67(1–2), 51–76.

- Hart, J., Gunnar, M. R., & Cicchetti, D. (1995). Salivary cortisol in maltreated children: Evidence of relations between neuroendocrine activity and social competence. *Development and Psychopathology*, 7, 11–26.
- Hennessy, M. B., Deak, T., & Schiml-Webb, P. A. (2001). Stress-induced sickness behaviors: An alternative hypothesis for responses during maternal separation. *Developmental Psychobiology*, 39(2), 76–83.
- Hennessy, M. B., O'Leary, S. K., Hawke, J. L., & Wilson, S. E. (2002). Social influences on cortisol and behavioral responses of preweaning, periadolescent, and adult guinea pigs. *Physiology & Behavior*, 76, 305–314.
- Henry, J. P. (1992). Biological basis of the stress response. *Integrative Physiological and Behavioral Science*, 27, 66–83.
- Hewig, J., Hagemann, D., Seifert, J., Naumann, E., & Bartussek, D. (2004). On the selective relation of frontal cortical asymmetry and anger-out versus anger-control. *Journal of Personality & Social Psychology*, 87(6), 926–939.
- Hofheimer, J. A., Wood, B. R., Porges, S. W., Pearson, E., & Lawson, E. E. (1995). Respiratory sinus arrhythmia and social interaction patterns in preterm newborns. *Infant Behavior and Development*, 18, 233–245.
- Huot, R. L., Brennan, P. A., Stowe, Z. N., Plotsky, P. M., & Walker, E. F. (2004). Negative affect in offspring of depressed mothers is predicted by infant cortisol levels at 6 months and maternal depression during pregnancy but not postpartum. *Annals of the New York Academy of Sciences 1. Biobehavioral Stress Response: Protective and Damaging Effects*, 1032, 234–236.
- Izard, C. E. (1977). *Human emotions*. New York: Plenum Press.
- Izard, C. E., Hembree, E. A., & Heubner, R. R. (1987). Infants' emotional expressions to acute pain: Developmental change and stability of individual differences. *Developmental Psychology*, 23, 105–113.
- Jennings, K. D. (2004). Development of goal-directed behaviour and related self-processes in toddlers. *International Journal of Behavioral Development*, 28(4), 319–327.
- Kagan, J. (1994). *Galen's prophecy*. New York: Basic Books.
- Kagan, J., Reznick, J. S., & Snidman, N. (1987). The physiology and psychology of behavioral inhibition in children. *Child Development*, 58, 1459–1473.
- Kerr, M., Tremblay, R., Pagani, L., & Vitaro, F. (1997). Boys' behavioral inhibition and the risk of later delinquency. *Archives of General Psychiatry*, 54(9), 809–816.
- Kohut, H. (1972). Thoughts on narcissism and narcissistic rage. *The Psychoanalytic Study of the Child*, 27, 360–400.
- Lerner, J. S., Gonzalez, R. M., Dahl, R. E., Hariri, A. R., & Taylor, S. E. (2005). Facial expressions of emotion reveal neuroendocrine and cardiovascular stress responses. *Biological Psychiatry*, 58, 743–750.
- Levine, S., Coe, C., & Weiner, S. G. (1989). Psychoneuroendocrinology of stress: A psychobiological perspective. In F. R. Bush & S. Levine (Eds.), *Psychoendocrinology* (pp. 341–377). New York: Academic Press.
- Lewis, M. (1990). The development of intentionality and the role of consciousness. *Psychological Inquiry*, 1(3), 231–247.
- Lewis, M. (1992). *Shame: The exposed self*. New York: Free Press.
- Lewis, M. (1993). The development of anger and rage. In R. A. Glick & S. P. Roose (Eds.), *Rage, power, and aggression: The role of affect in motivation, development and adaptation*. New Haven, CT: Yale University Press.
- Lewis, M. (1995). *Shame: The exposed self (Paperback edition)*. New York: The Free Press.
- Lewis, M., Alessandri, S. M., & Sullivan, M. W. (1990). Violation of expectancy, loss of control, and anger expressions in young infants. *Developmental Psychology*, 26(5), 745–751.
- Lewis, M., & Goldberg, S. (1969). Perceptual-cognitive development in infancy: A generalized expectancy model as a function of mother-infant interaction. *Merrill-Palmer Quarterly*, 15, 81–100.
- Lewis, M., Hitchcock, D. F. A., & Sullivan, M. W. (2004). Physiological and emotional reactivity to learning and frustration. *Infancy*, 6(1), 121–143.
- Lewis, M., & Michalson, L. (1983). *Children's emotions and moods: Developmental theory and measurement*. New York: Plenum Press.
- Lewis, M., & Ramsay, D. (2005). Infant emotional and cortisol responses to goal blockage. *Child Development*, 76, 518–530.
- Lewis, M., Ramsay, D., & Sullivan, M. W. (2006). The relation of ans and hpa activation to infant anger and sadness response to goal blockage. *Developmental Psychobiology*, 48, 397–405.
- Lewis, M., Sullivan, M. W., & Michalson, L. (1984). The cognitive-emotional fugue. In J. Kagan, C. E. Izard, & R. B. Zajonc (Eds.), *Emotions, cognitions, and behavior* (pp. 264–288). London: Cambridge University Press.
- Lewis, M., Sullivan, M. W., Ramsay, D., & Alessandri, S. M. (1992). Individual differences in anger and sad expressions during extinction: Antecedents and consequences. *Infant Behavior & Development*, 15(4), 443–452.
- Liu, D., Diorio, J., Tannenbaum, B., Caldji, C., Freedman, F. D., Sharma, S., et al. (1997). Maternal care, hippocampal glucocorticoid receptors, and hypothalamic-pituitary-adrenal responses to stress. *Science*, 277, 1659–1662.

- Lundberg, U., Rasch, B., & Westermarck, O. (1990). Familial similarity in Type A behavior and physiological measurements. *Scandinavian Journal of Psychology*, 31(1), 34–41.
- Lutkenhaus, P. (1984). Pleasure derived from mastery in 3-year-olds: Its function for persistence and the influence of maternal behavior. *International Journal of Behavioral Development*, 7, 343–358.
- MacTurk, R. B., & Morgan, G. A. (Eds.). (1995). *Mastery motivation: Origins, conceptualizations, and applications*. Norwood, NJ: Ablex.
- Malatesta, C. Z., & Haviland, J. (1982). Learning display rules: The socialization of emotional expression in infancy. *Child Development*, 53, 991–1003.
- Marquis, D. (1943). A study of frustration in newborn infants. *Journal of Experimental Psychology*, 23, 123–138.
- Matthews, K. A., Woodall, K. L., Engebretson, T. O., & McCann, B. S. (1992). Influence of age, sex and family on type-A and hostile attitudes and behaviors. *Health Psychology*, 11(5), 317–323.
- Messer, D. (Ed.). (1993). *Mastery motivation in early childhood: Development, measurement, and social processes*. London: Routledge.
- Moore, G. A., & Calkins, S. D. (2004). Infants' vagal regulation in the still-face paradigm is related to dyadic coordination of mother-infant interaction. *Developmental Psychology*, 40(6), 1068–1080.
- Morrison, A. P. (Ed.). (1986). *Essential papers on narcissism*. New York: New York University Press.
- Nachimas, M., Gunnar, M. R., Mangelsdorf, S., Parritz, R. H., & Buss, K. (1996). Behavioral inhibition and stress reactivity: The moderating role of attachment security. *Child Development*, 67, 508–522.
- Nietzsche, F. ([1904]1964). *Will to power*. New York: Russell & Russell.
- Oginska-Bulik, N., & Juczynski, Z. (1998). Psychological correlates of the type A behavior pattern in children. *Polish Psychological Bulletin*, 29(4), 313–319.
- Panksepp, J. (1998). *Affective neuroscience*. New York: Oxford Press.
- Piaget, J. (1952). *The origins of intelligence in children*. New York: International Universities Press.
- Plutchik, R. (1980). A general psychoevolutionary theory of emotion. In R. Plutchik & H. Kellerman (Eds.), *Emotion: Theory, research, and experience* (Vol. 1, pp. 3–33). New York: Academic Press.
- Porges, S. W. (1998). Love and the evolution of the autonomic nervous system: The polyvagal theory of intimacy. *Psychoneuroendocrinology*, 23, 837–861.
- Porges, S. W. (2001). The polyvagal theory: Phylogenetic substrates of a social nervous system. *International Journal of Psychophysiology*, 42(2), 123–126.
- Porges, S. W., Doussard-Roosevelt, F. A., Portales, L., & Greenspan, S. (1996). Infant regulation of the vagal “brake” predicts child behavioral problems: A psychobiological model of social behavior. *Developmental Psychology*, 29, 697–712.
- Potegal, M., Robison, S., Anderson, F., Jordan, C., & Shapiro, E. (2007). Sequence and priming in 15 month olds' reactions to brief arm restraint: Evidence for a hierarchy of anger responses. *Aggressive Behavior*, 33, 1–11.
- Putnam, S. P., & Stifter, C. (2005). Behavioral approach-inhibition in toddlers: Prediction from infancy, positive and negative affective components, and relations with behavior problems. *Child Development*, 76(1), 212–226.
- Ramsay, D., King, S., & Lewis, M. (2005). The role of infant emotional response to goal blockage on toddler persistence. Poster presented at the SRCB Biennial International Conference, Atlanta, GA, April 7–10.
- Rank, O. (1945). *Will therapy and truth and reality*. New York: Knopf.
- Richards, J. E. (1995). Reliability of respiratory sinus arrhythmia in r-r intervals in 14-, 20-, and 26-week-old infants. *Infant Behavior and Development*, 18, 155–161.
- Rothbaum, F., Wolfer, J., & Visintainer, M. (1980). Coping behavior and locus of control in children. *Journal of Personality*, 47, 118–135.
- Rubin, K. H., Hastings, P. D., Stewart, S. L., Henderson, H. A., & Chen, X. (1997). The consistency and concomitants of inhibition: Some of the children, all of the time. *Child Development*, 68, 467–483.
- Sahar, T., Shalev, A. Y., & Porges, S. W. (2001). Vagal modulation of responses to mental challenge in post-traumatic stress disorder. *Biological Psychiatry*, 47(7), 637–643.
- Sallis, J. F., Dimsdale, J. E., & Chipman, C. (1988). Invited review: Blood pressure reactivity in children. *Journal of Psychosomatic Research*, 32(1), 1–12.
- Schmidt, L. A., Fox, N., Rubin, K. H., Sternberg, E. M., Gold, P. W., Smith, C. C., et al. (1997). Behavioral and neuroendocrine responses of shy children. *Developmental Psychobiology*, 30, 127–140.
- Schneirla, T. C. (1959). An evolutionary and developmental theory of biphasic processes underlying approach and withdrawal. In M. R. Jones (Ed.), *Nebraska symposium on motivation* (Vol. 7, pp. 1–42). Lincoln: University of Nebraska Press.
- Sears, R., & Sears, P. (1940). Minor studies of aggression, V: Strength of frustration-reaction as a function of strength of drive. *Journal of Psychology*, 9, 297–300.
- Seligman, M. E. P. (1975a). *Helplessness: On depression, development and death*. San Francisco: Freeman.

- Seligman, M. E. P. (1975b). *On depression, development, and death*. San Francisco: W. H. Freeman.
- Seligman, M. E. P. (1991). *Learned optimism*. New York: Knopf.
- Sherman, M., & Sherman, I. C. (1925). Sensorimotor responses in infants. *Journal of Comparative Psychology*, 5, 53–68.
- Sherman, M., Sherman, I. C., & Flory, C. D. (1936). Infant behavior. *Comparative Psychology Monographs*, 12, 1–107.
- Solomon, R. L. (1977). An opponent process theory of acquired motivation: The affective dynamics of addiction. In J. D. Maser & M. E. P. Seligman (Eds.), *Psychopathology: Experimental models* (pp. 66–103). San Francisco: W. H. Freeman.
- Spangler, G., & Grossman, K. E. (1993). Behavioral organization in securely and insecurely attached infants. *Child Development*, 64, 1439–1450.
- Spangler, G., Schieche, M., Lllg, U., Maier, U., & Ackerman, C. (1994). Maternal sensitivity as an external organizer for biobehavioral regulation in infancy. *Developmental Psychobiology*, 27, 425–437.
- Sroufe, L. A. (1996). *Emotional development: The organization of emotional life in the early years*. New York: Cambridge University Press.
- Stein, N. L., & Jewett, J. L. (1986). A conceptual analysis of the meaning of negative emotions: Implications for a theory of development. In C. E. Izard & P. B. Read (Eds.), *Measuring emotions in infants and children* (Vol. 2, pp. 238–267). New York: Cambridge University Press.
- Stenberg, C. R. (1982). *The development of anger facial expressions in infancy*. Doctoral dissertation, University of Denver, Colorado.
- Stenberg, C. R., & Campos, J. J. (1990). The development of anger expressions in infancy. In N. L. Stein, B. Levanthal, & T. Trabasso (Eds.), *Psychological and psychobiological approaches to emotion* (pp. 247–267). Hillsdale, NJ: Erlbaum.
- Stenberg, C. R., Campos, J. J., & Emde, R. N. (1983). The facial expression of anger in seven-month-old infants. *Child Development*, 54, 178–184.
- Suarez, E. C., Williams, R. B., Kuhn, C. M., Zimmerman, E., & Schanberg, S. M. (1991). Biobehavioral basis of coronary-prone behavior in middle-aged men, part ii: Serum cholesterol, type-a behavior pattern, and hostility as interactive modulators of physiological reactivity. *Psychosomatic Medicine*, 53, 528–537.
- Sullivan, M. W. (2007). *Individual differences in how babies become frustrated toddlers*. Paper presented at the Institute for the Study of Child Development Brown Bag Series, UMDNJ-Robert Wood Johnson Medical School, New Brunswick, NJ, October 25, 2007.
- Sullivan, M. W., & Lewis, M. (2003). Contextual determinants of anger and other negative expressions in young infants. *Developmental Psychology*, 39(4), 693–705.
- Sullivan, M. W., Lewis, M., & Alessandri, S. M. (1992). Cross-age stability in emotional expressions during learning and extinction. *Developmental Psychology*, 28(1), 58–63.
- Suomi, S. J., Mineka, S., & Harlow, H. H. (1983). Social separation in monkeys as viewed from several motivational perspectives. In E. Satinoff & P. Titelbaum (Eds.), *Handbook of neurobiology: Motivation* (Vol. 6). New York: Plenum.
- Tronick, E. (1989). Emotions and emotional communication in infants. *American Psychologist*, 44, 112–119.
- Turner, L. A., & Johnson, M. C. (2003). A model of mastery motivation for at-risk preschoolers. *Educational Psychology*, 95, 495–505.
- Wacker, J., Heldmann, M., & Stemmler, G. (2003). Separating emotion and motivational direction in fear and anger: Effects on frontal asymmetry. *Emotion*, 3(2), 167–193.
- Waldstein, S. R., Kop, W. J., Schmidt, L. A., Haufier, A. J., Krantz, D. S., & Fox, N. (2000). Frontal electrocortical and cardiovascular reactivity during happiness and anger. *Biological Psychology*, 55(3–23).
- Watson, J. B. (1925). Experimental studies on the growth of the emotions. *Pediatric Seminars*, 32, 328–348.
- Watson, J. S. (1972). Smiling, cooing, and the game. *Merrill-Palmer Quarterly*, 18, 323–339.
- White, R. W. (1959). Motivation reconsidered: The concept of competence. *Psychological Review*, 66, 297–323.
- van Bakel, H. J., & Riksen-Walraven, J. M. (2004). Stress reactivity in 15-month old infants: Links with infant temperament, cognitive competence, and attachment security. *Developmental Psychobiology*, 44(3), 157–167.

Chapter 12

Anger in Children's Tantrums: A New, Quantitative, Behaviorally Based Model

Michael Potegal and Peihua Qiu

Abstract Because excessive anger in early childhood can predict later psychopathology, quantifying its intensity and time course is clinically important. Anger consists of a set of experiential, physiological, and behavioral responses whose coherence is sufficient to justify the assumption of a common latent variable that can vary in intensity. The relationships between anger intensity and various anger-driven behaviors in children's tantrums are probabilistic, nonlinear, and different for each individual behavior. Although any one behavior can provide only a partial and indirect measure of anger intensity, the entire trajectory of anger across the tantrum may be reconstructed by combining the observed temporal distributions of the various behaviors. In particular, we observed that behaviors characteristic of lower intensities of anger tend to occur at both the beginning and the end of tantrums while behaviors linked to higher intensities of anger are distributed around a single early peak. Accordingly, our anger intensity-behavioral linkage function model reconstructs a single, common, latent anger intensity variable, $MA(t)$, whose rise and fall controls the momentary probability of eight angry tantrum behaviors through linkage functions that are unique to each behavior. We introduce the MA50 as a practical measure of the "characteristic" intensity of the eight angry behaviors and note how the model may inform study of the neural substrates of anger.

12.1 Introduction and Chapter Organization

This chapter is organized as follows: Section 12.2 reviews the behavioral phenomenology and clinical importance of childhood anger, the value of quantifying its intensity and time course, and some of the difficulties involved in doing so. We argue that anger consists of a set of experiential, physiological, and behavioral responses whose coherence is sufficient to justify the assumption of a common latent variable that can vary in intensity. We note that although behaviors (or "action tendencies") are particularly salient indicators of anger, the relationships between anger intensity and various behaviors are probabilistic (rather than deterministic), nonlinear (because different behaviors become most probable within different portions of the anger intensity range), and different for each individual behavior. Thus, each behavior can provide only a partial and indirect measure of

M. Potegal (✉)
University of Minnesota, Minneapolis, MN, USA
e-mail: poteg001@umn.edu

anger intensity. However, the observation that different behaviors are characteristic of different portions of the anger intensity range implies that the entire trajectory of anger may be reconstructed by combining the temporal distributions of the various behaviors. The model presented in Section 12.3 accomplishes this through a single, common, latent anger intensity variable, $MA(t)$, whose rise and fall controls the momentary probability of eight angry tantrum behaviors through linkage functions that are unique to each behavior. This model is predicated upon two basic observations. First, anger is episodic and, within an episode, anger intensity rises and falls over time. Second, some behaviors are characteristic of lower intensities of anger while other behaviors are more closely linked to higher intensities of anger. From these two observations, it follows that “low anger” behaviors should be more likely toward the beginning and the end of an episode, when anger intensity is lower, while “high anger” behaviors are most likely to be distributed around a single point within the episode, when anger is peaking. The observation of just such unimodal distribution of high anger behaviors whose peak coincided with the trough in the bimodal distribution of low anger behaviors in children’s tantrums (Potegal, Kosorok, & Davidson, 2003, Fig. 12.3) was the inspiration for our model. Section 12.3 also introduces the MA50 as a practical measure of the “characteristic” intensity of the eight angry behaviors. Sections 12.4, 12.5, and 12.6 note current limitations and future directions for model development, describe extensions and applications to different measures and groups, and review applications to, and implications for, appraisal models of emotion and emotion display rules. Section 12.7 notes how the model may inform study of the neural substrates of anger.

12.2 Phenomenology and Importance of Childhood Anger

Episodes of anger are frequent in early childhood. Young children may express their anger first by grunting and growling; they may then escalate to shouting and screaming (Potegal & Davidson, 2003). Children may grab, push, or pull; becoming more angry, they hit and kick. Some run away (Eisenberg et al., 1999); others throw their heads back and become so rigid with tension that they suffer painful, involuntary muscle spasms of the fingers and toes. These responses are surprising in their diversity and can be striking in their intensity; the more extreme forms can be alarming to the parents who witness them. Beyond these dramatic displays, the reasons for focusing on anger in development include the observation that excessive anger at 2 years of age predicts increased risk of psychopathology at 5–6 (Radke-Yarrow & Kochanska, 1990). In turn, excessive anger at 4–6 years of age predicts socially inappropriate behavior in school (e.g., low social skills and excessive aggression), and behavior problems at home, through 8–10 years (Eisenberg et al., 1999). Anger is especially potent in exacerbating aggression in 7–13 year olds boys with behavior problems (Orobio de Castro, et al., 2005). Excessively intense and prolonged episodes of anger (“severe rages”) have been suggested as a diagnostic criterion for the “broad phenotype” of juvenile onset bipolar disorder (Leibenluft, Charney, Towbin, Bhangoo, & Pine, 2003, but see Potegal, Carlson et al., 2009). A reliable measure of anger is needed to answer questions such as Is a child’s anger and anger regulation (in) appropriate for her age? How, exactly, is anger related to risk of clinical impairment? How successful is a given treatment in fostering control and reducing or limiting anger?

12.2.1 Rationale for Quantifying Anger

Progress in science depends upon quantification, but little is known about quantifying anger (e.g., Fridja et al., 1992). In the past, emotion research has sometimes focused more on words relating to anger rather than on behaviors associated with it. In this tradition, some authors have suggested

that the term “anger” reflects a level of intensity intermediate between lower level “annoyance” and higher level “rage” (e.g., Plutchik, 1980). By having subjects rate their own individual experiences on 1–10 scales using different anger-related words, Fridja et al. (1992) verified the intuition that words in the English lexicon ranging from “irritated” or “annoyed” through “angry” to “furious” or “enraged” represent an internally consistent dimension of anger intensity. To the notion that anger intensity varies along continuum from annoyance and irritation up to rage and fury, Lewis (this book) adds that rage is distinguished from anger by behavior that is not goal directed and that is out of control.

12.2.2 Anger as a Coupled Response System

There is a general consensus that anger, like other emotions, is a complex system of responses that are variably coupled (e.g., Gross, 1998). These responses include appraisal processes and cognitions, subjective feelings, physiological arousal, facial and vocal expressions, and certain acts and/or impulses to action (e.g., to hit or to hurt, depending on developmental level, Freud (1972), Feshbach, 1964). Each of these responses has some previous or potential use in quantifying anger intensity and all should eventually be included in a quantitative theory of anger. Up to now, subjective self-report and facial expressions have been the primary measures. However, there are theoretical and methodological issues with these measures, which we review briefly.

12.2.3 Current Measures of Anger Intensity

Even today, in the twenty-first century, psychologists have had to rely upon subjective self-reports along arbitrary numerical scales to estimate anger (e.g., Hoeksma, Oosterlaan, & Schipper, 2004). Such reports have the virtue of being easy to elicit. However, establishing their reliability remains a challenge, especially for young children who are notoriously poor reporters of their own anger (e.g., Dearing et al., 2002; Denham & Couchoud, 1990; Levine, Stein, & Liwag, 1999).

As reviewed by Green et al. and Matsumoto et al., respectively (this book), vocal and facial expressions are among the most salient objective indicators of anger. Vocal anger in adults is characterized by increases in tempo and co-varying increases in loudness and pitch; these effects can be readily distinguished from those associated with, e.g., fear or sadness (Green et al., this book.) Although the recognition of affective state from voice samples is considerably better than chance and is as good as, or better than, from facial expression, the most extensively studied objective measures of anger are, indeed, facial expressions. These involve lowering of the brows, narrowing of the palpebral fissure, and increased tension around the mouth (e.g., Matsumoto et al., this book). Facial expressions have much to recommend them as a quantitative measure. The “universality” claim, that expressions of anger (like the other basic emotions) are similar across cultures, is still debated (e.g., Russell, 1997), but is supported by the most recent and rigorous cross-cultural studies (Chapter 8 by D. Matsumoto et al., this volume). Although anger-related facial action units (AU) were once thought to be more difficult to recognize or label (Ekman, 1994, c.f., Russell, 1995), expressions of anger were among the facial expression most consistently recognized cross-culturally, with correct identification in the 80–90% range (Haidt & Keltner, 1999). The total number of co-occurring AUs marks the level of self-reported anger (Alvarado & Jameson, 2002); thus, facial expression can be used to scale anger intensity. Methodologically, raters can estimate the intensity of facial anger reliably (e.g., Hess, Blairy, & Kleck, 1997; Matsumoto, 1989).

12.2.4 *Coherence of Measures*

There are, however, limitations on facial expressions as a measure of anger. Facial displays of anger are actually relatively rare in adults, even in experimental situations designed to provoke anger. This discrepancy is one instance of a lack of agreement, or “coherence”, among facial expressions of emotions, self-reported affect, and other measures. Such coherence is often assumed, and some experimental evidence supports it (e.g., Matsumoto, Nezlek, & Koopmann, 2007), but serious criticisms have been raised with regard to coherence, at least for some emotions (e.g., Fernandez-Dols, Sanchez, Carrera, & Ruiz-Belda, 1997). For anger, however, three of four studies have found substantial coherence of measures in adults, at least under some conditions. Significant correlations between facial expressions and reported feelings of anger were found in a posed expression task (Coan & Allen, 2003), in a more naturalistic interview study of conjugal bereavement when people were talking about the injustice of their spouse’s death ($r = 0.44$, Bonano & Keltner, 2004), and when mothers interacted with their preschool daughters in an experimental frustration task ($0.42 \leq r \leq 0.57$, Cole, Teti, & Zahn-Waxler, 2003). Coherence of emotion measures is thought to be greater at higher intensities of emotion (e.g., Fridja et al., 1992; Davidson, 1992; Rosenberg & Ekman, 1994; Tassinanry & Cacioppo, 1992). However, although facial expressions of anger distinguished subjects in Stemmler’s (1997) study who reported having been slightly angered by exposure to the mild variant of an insult manipulation from those who had been exposed to the moderate or “full” anger variants, facial expressions did not distinguish the moderately from the “fully” angered subjects. That is, facial expression failed to distinguish anger intensity at the upper end of the adult range.

Coherence may be stronger in children. This conclusion is suggested by three studies of 7–12 year olds who were confronted by an anger-provoking peer confederate, either live or on videotape. Children who lost a computer game and were taunted by the winner (Underwood & Bjornstad, 2001) had angry facial expressions that were mildly but significantly correlated with the children’s report of being bothered by the winner’s taunts ($r = 0.18$). There were some curious inversions of expected responses reported in this study, with mad feelings being associated with distress gestures (hanging the head, covering the face to cry, or crying) and sad feelings being associated with angry gestures (hostile stares and glares, flinching in exasperation, banging the keys in frustration, invading the other’s personal space, and banging against the actor’s chair). Greater coherence was found in responses to peer disapproval, e.g., self-reported anger was significantly associated with negative facial expressions including anger, contempt, disgust, fear, and sadness. These effects were particularly marked among girls and 12 year olds ($0.39 \leq r \leq 0.6$ Casey, 1993). Coherence was the greatest and most specific in children who lost a competitive game to a peer confederate who cheated (Dearing et al., 2002; Hubbard et al., 2004). There were significant correlations among six of the ten pairs of measures that included facial expression and self-report of expressed anger. Notably, angry behaviors (e.g., throwing or slamming down game pieces, swinging a fist or punching it into the opposite hand, hitting their own head) were correlated with the largest number of other measures including facial expressions, self-report, and skin conductance reactivity. These effects were progressively stronger for children judged to show low, average, or high-reactive aggression. In fact, these angry behaviors were the only measures that significantly predicted the child’s judged aggressiveness and his/her social rejection in peer sociometric ratings.

Beyond the coherence issue, the theoretical basis for using facial expressions to measure anger is challenged by the functionalist caveat that facial expressions are a means of communication and/or social manipulation (e.g., Fridlund, 1997). From at least the age of 4 or 5, facial expressions of anger appear to be a mixed signal, influenced not only by the individual’s internal state, but by

her communication goals as well (see “Display rules,” Section 5.4). Methodologically, studies that capture facial expressions on film or videotape are most often carried out under restricted laboratory conditions. The dynamic range of facial expressions in naturalistic situations is unknown, but they may not reliably reflect affect at its extremes. At the lower end of the intensity range, 15 month olds respond to brief, gentle arm restraint with a highly systematic progression of responses (Potegal et al., 2007). They first struggled against the restraint and then protested vocally. Angry facial expressions appeared last. This progression of behaviors was smooth and overlapping, i.e., the earlier behaviors continued even after the later behaviors began. Thus, this progression appeared to reflect a steadily increasing intensity of anger in which the lower intensities were not reflected in facial expressions. At the upper end of the range, Stemmler's (1997) results suggest that angry expressions do not become progressively more pronounced as anger rises from baseline all the way to its peak but that they saturate at some sub-maximal level. While facial expressions are surely valuable indicators within some range of anger intensities, it is unlikely that they are sensitive indicators throughout the full range.

12.2.5 Behavioral Measures of Anger Intensity

In everyday life, we detect the anger of others by an array of behavioral and physiological cues: facial expression, loudness, pitch and tempo of voice, facial flushing, threatening or aggressive actions, and so forth. In both infants (Camras, Sullivan, & Michel, 1993) and adults (Wallbott, 1998), anger can be reliably differentiated from sadness (and some other emotions) by the greater amplitude and “jerkiness” of its associated body movements. Although some people's anger-associated behaviors may be subtle and/or idiosyncratic, we are often correct in gauging the intensity of children's anger by their behavior; a grimace and a grunt may indicate their irritation; a shout, their anger; a screaming, hitting and kicking attack, their rage. Accordingly, gestural expressions were the strongest or most general indicator of children's anger in at least two of the studies above (Underwood & Bjornstad, 2001; Hubbard et al., 2004). These observations further imply that each angry behavior may reflect a “characteristic” range of anger intensity. In keeping with these objective observations of behavior, an analysis of self-reports by adults suggests that emotions can be more readily distinguished from each other by their action tendencies than by their feeling states; the action tendencies reported for anger were yelling and hitting (Roseman, Wiest, & Swartz, 1994). Etymologically, the Greek (anchein) and Latin (angor) roots of the word anger refer to “strangling.” Adults' propensity for angry actions tends to fall along a single, “Anger Out” scale which has been made psychometrically sound by Spielberger and colleagues within their frequently used *State-Trait Anger Expression Inventory* (Chapter 23 by C.D. Spielberger and E.C. Reheiser, this volume). In expanding the items on the Anger Out scale, Deffenbacher, Oetting, Lynch, and Morris (1996) identified two clusters of verbal anger expression (loud and argumentative, intimidating and threatening) and two clusters of physical anger expression (threatened or actual assault, property damage). Although separable, these clusters were correlated with each other and with trait anger, again suggesting that these overt behaviors all reflect the same or related underlying process(es). Furthermore, these behaviors are associated with the subjective experience of anger. In both hypothetical scenarios (Winstok, 2007) and recalled incidents of actual everyday emotion (Sonnemans & Frijda, 1994), the reported “drasticness” of action was one of the strongest predictors of overall intensity of felt anger (c.f., Shaver, Wu, & Schwartz, 1992). The continuity between feelings of anger and angry behavior, and their scalability, was shown in a three-item scale of increasing anger intensity (feeling annoyed, feeling angry, and yelling at someone) developed by Ross and colleagues (Mirowsky & Ross, 1995; Ross &

Van Willigen, 1996, Scheiman, this book.) This scale was found to have Guttman scale-like transitivity, i.e., across subjects, yelling represented higher intensity than feeling angry. In turn, feeling angry represented higher intensity than just feeling annoyed.

Quantifying the intensity of anger relative to a range of identifiable behaviors would be scientifically useful, not only in designing new studies, but in interpreting older ones. One costly, large scale, longitudinal study of children's anger in this context recorded "pushes and shoves" (Huesmann, Eron, Lefkowitz, & Walder, 1984); other such studies carefully document the emergence of "hits, kicks, and bites" (e.g., Nagin & Tremblay, 2001). Because there has been no way to scale the intensity of children's various angry behaviors to produce a rational estimate of their overall anger, these worthwhile studies are more difficult to compare.

12.2.6 The Time Course of Anger

Like other emotions, anger rises, then falls in the course of a typical episode. As reviewed by Potegal (this book), the rise is typically rapid and the fall slower. Other than this, little is known about the trajectory of anger. Really interesting questions about anger dynamics remain to be asked: How do shorter and longer episodes of anger differ? Do these episodes begin similarly, but then differ because anger continues to increase to a higher level in what will become the longer events? If, on the other hand, shorter and longer events differ systematically from onset, is a more rapid rise of anger associated with a shorter or a longer event, a lower or higher peak?

12.2.7 Challenges and Opportunities for Behavioral Quantification of Anger

While a rating scale for anger intensity based on observable behaviors has the obvious advantage that behaviors can be recorded and coded with great reliability, the behaviors themselves are topographically and physiologically diverse. For example, Eckhardt and Deffenbacher's (1995) list of nine categories of anger-related behaviors among adults includes sullen withdrawal and "icy stares"; refusal to cooperate with others; sarcasm, hostile humor, and cutting remarks; verbal threats; and various forms of physical aggression against property and people. The angry behaviors we commonly observe in others, and experience in ourselves, are not only highly diverse, they also appear discontinuous within episodes. Thus, when we adults are mildly angry, we may cross our arms and purse our lips. As we become more angry, we do not cross our arms more tightly harder or purse our lips harder. Instead, we may retract our lips in a grimace, grit our teeth, or wag a finger, and then move up to swearing and shouting and eventually, perhaps, to physical assault. That some behaviors are more probable at low anger intensity while others become more probable at higher intensities suggests the possibility that behaviors can be ordered or weighted by their "characteristic intensity" of anger. Furthermore, the common experience that the intensity of a person's anger at a given point in time can often be correctly inferred from his/her behaviors at that point implies that the set of these behaviors may together span the intensity range. In turn, this suggests the possibility of reconstructing both the overall trajectory of anger and the functions linking the probability of each behavior to anger from the observed distributions of these behaviors within anger episodes. However, we adults tend to mask emotions; our angry responses are also tempered by our status relative to the offender (Kuppens, Mechelen, & Van Meulders, 2004), by fear of retaliation (Winstok, 2007), and so forth. These concerns, as well as individual idiosyncrasies in adults' expression of anger, present a serious problem for modeling.

12.2.8 Temper Tantrums: A Solution to the Problem

To define the trajectory of anger and find its links to individual angry behaviors from an observed distribution of behaviors, it is necessary to have a substantial database in which a relatively small number of stereotyped, easily classifiable angry behaviors appear with some frequency. Not having fully acquired social display rules, young children are less likely to mask their emotions than adults. In particular, temper tantrums are common in children between the ages of 18 and 60 months, many of whom have them up to once or twice per day (Potegal & Archer, 2004). Tantrums typically occur in the familiar home environment among people with whom children feel comfortable (Einon & Potegal, 1994), so emotional expression is likely to be uninhibited. Unlike the more idiosyncratic anger of adults, angry behaviors within tantrums are both similar and common enough across children to be amenable to study. Also unlike adults' naturalistic anger episodes that can involve two or more participants whose interactions are difficult to control, the role of parents in tantrums can, to some extent, be reduced and standardized.

It is a truism that there are no individual behaviors that are either necessary or sufficient for defining an episode of anger. In fact, each of the angry behaviors in a tantrum can occur by itself, although the base rates for such behaviors outside a tantrum are low (e.g., Snyder, Stoolmiller, Wilson, & Yamamoto, 2003). However, when several of them co-occur in a short period with obviously increased intensity, changed rhythm and forcefulness of vocalization and movement, in addition to physiological signs such as facial flushing (i.e., when all anger response systems are activated), there is little doubt that the child is angry. Conversely, it is also generally clear when a child is faking it.

12.3 Modeling Methods, Data, and Results

Here, we first describe two tantrum data sets collected with different techniques at different times and places, but whose intensity-related groupings of angry behaviors are similar, nonetheless. We then describe the anger intensity-behavioral linkage function model.

12.3.1 The Data

The first data set consisted of retrospective written narratives collected in 1993–1995 from parents in Madison, WI area who described one of their child's tantrums in detail (Potegal & Davidson, 2003; Potegal, Kosorok, & Davidson, 2003). The current analysis includes a total of 127 of these tantrums had by those 3- and 4-year olds (65 boys, 62 girls) in which at least one angry behavior occurred. For details of data reduction, tantrum reconstruction, etc., see Potegal and Davidson (2003), Potegal et al. (2003). Children in the first and second sets were recruited largely from volunteer lists maintained by the University of Wisconsin's Waisman Center and the University of Minnesota's Institute of Child Development, respectively. Most were white and middle class. The second set of 119 in-home tantrums had by 59 of 3- and 4-year olds (41 boys, 18 girls) was collected in the Minneapolis, MN area in 2001–2003 (Potegal, 2003, 2005). Each child contributed up to three tantrums to this latter data set. A more rigorous methodology was used to collect these data. In brief, tantrum behaviors were recorded by parents on a user-friendly coding form with "anatomical" ordering of child behaviors into six rows to make them easy to find. For example, tears were noted in a top row with a "face" icon, a "voice" row for vocalizations was just below,

and a row with a leg icon for kicking was near the bottom. Parents recorded behaviors in four consecutively labeled 30 s columns across each page with the help of a purpose-built coding timer (Advanced Research Corporation, Minneapolis, MN). The timer's timing circuit successively illuminated LEDs mounted above each of the coding columns on the form, thus directing parent's attention to the column to be used at the moment. The timer also contained an audio-cassette recorder that recorded vocalizations directly; the timing circuit placed a marker signal on the tape at 30 s intervals for subsequent synchronization with parental observations. During a 2 h home visit, parents learned to use the timer by coding a videotape containing four "composite" tantrums (i.e., compiled and edited clips of real tantrums). Reliabilities for training tape coding were reasonable; mean kappas for parents' coding of behaviors classified as low, intermediate, and high anger and distress (Table 12.1) were 0.84, 0.91, 0.69, and 0.73, respectively. Using the timer also limited parent mobility, thereby tending to minimize physical intervention in the tantrum (parents were instructed to abandon coding of any tantrum that required extensive intervention).

In all cases, tantrums were reconstructed from the records taking the first recorded behavior as the start point of the tantrum. In the WI study, all behaviors were coded from the parent narratives. In the MN studies, physical behaviors were taken from the parental coding form while trained raters coded vocalizations (anger-related shouting and screaming and distress-related whining and crying) from the audiotape. For the latter coding, shout was defined as a loud vocalization, usually containing words; scream was defined as a higher pitched vocalization, usually without words. Inter-rater reliabilities (kappa coefficients) for screaming, shouting, whining, and crying were 0.74, 0.72, 0.65, and 0.83, respectively.

12.3.2 Principal Components Analyses

Separate principal components analyses of $\log(x+1)$ transformed cumulative behavior durations in the two studies produced highly similar solutions. Each analysis yielded three components that appear to reflect different intensities of anger, and one component of distress. In both analyses, the order of eigenvalues was the same and the components together accounted for >50% of the variance. Furthermore, as shown by the main diagonal entries in Table 12.1, most behaviors load on the same components in both analyses. *Kick*, *scream*, and *arch* load on a principal component named "high anger", *throw* loads on "intermediate anger", and *stamp* loads on "low anger." *Hit* loads on both high and low anger in each analysis. Relatively minor differences between the two analyses include the shifts in *shout* and *push* to higher loadings on high and intermediate anger, respectively, and a shift of *run away* into intermediate anger. The major difference is that whine appears in distress in the WI data and in high anger in the MN data. This shift may be due to some co-variation across types of vocalization, i.e., whine, shout, and scream may tend to co-occur across children (Section 4.2.7)

In both data sets, the identification of the factors with different levels of anger intensity was supported by multiple regressions indicating that the high anger factor had higher correlations with tantrum duration, visible autonomic activation (e.g., tears, flushing), and parental judgment of overall tantrum severity than did the lower anger factors (Table 12.2). Thus, the fact that differences in the factor structure and behavior loadings are minor in the face of the major differences in sampling and data collection methodology speaks to the robust linkages between tantrum behaviors and anger intensity. Similar groupings of low vs. high intensity anger behaviors can be seen in Mascolo, Harkins, and Harakal (2000) clusters of "frustration" vs. "anger" related behaviors in preschoolers' social conflicts (e.g., *jump* and *throw* vs. *grab* and *scream*, respectively). Like these authors, we also

Table 12.1 Comparison of behavior loadings on principal components in WI and MN tantrums

Minnesota					
Principal components	High anger	Intermediate anger	Low anger	Distress	Eigen values
WISCONSIN	High anger				
	Kick	0.73/ 0.54	Push 0.39/ 0.69		2.03
	Scream	0.63/ 0.56			
	Arch	0.49/ 0.57			
	Hit	0.50/ 0.40			
	Shout	0.66/ 0.83	Throw 0.81/ 0.65		1.27
Intermediate anger					
Low anger					
			Stamp Flap	0.79/ 0.78 */ 0.68	1.15
Distress			Hit	0.39/ 0.47	
	Whine	0.58/ 0.68		Cry 0.67/ 0.58	1.46
Coping style				Seek comfort 0.67/ 0.82	
				Down 0.62/ 0.53	1.14
Eigen values	3.6	1.32	1.22	1.47	

Loadings on respective WI and MN tantrum components are shown as: *italic* / **bold**. The WI results are from Potegal and Davidson (2003), Table 4.
*Not included in WI narratives. “Down” indicates lowering the body. “Away” indicates moving or running away from parent.

Table 12.2 Regressions of WI and MN principal components on tantrum duration, autonomic activation and judged severity

Factor	Tantrum duration		Visible autonomic activation		Judged severity	
	WI	MN	WI	MN	WI	MN
High anger	0.39***	0.18***	0.31***	0.30**	0.25***	0.26**
Intermediate anger	0.26***	0.002	0.14**	−0.01	0.09	0.05
Low anger	0.03	0.09*	0.07	0.07	0.02	0.14
Distress	0.50***	0.78***	0.17**	−0.001	0.28***	0.16

* $p < 0.02$, ** $p < 0.01$, *** $p < 0.005$ WI results from Potegal and Davidson (2003), Table 5.

found distress (sadness) to be a separate factor in both data sets. The generality of this factor structure is not limited to children under 5; very similar factors were found in the outbursts of 5–12 year old inpatients on a child psychiatry ward (Potegal, Carlson, Margulies, Gutkovitch, & Wall, 2009).

12.3.3 The Model

The elements of the anger model are momentary anger [MA(t)], a single, latent variable whose trajectory controls the overall time course of the various angry behaviors, and a set of linkage functions which express the probability of each behavior as unique functions of MA. The two-parameter beta function was chosen to represent MA(t) because it can assume a variety of trajectories depending upon its parameters. Figure 12.1, which includes a monotonic rise, a monotonic fall, and also U and inverted U shapes with different skews, illustrates how the polymorphic flexibility of the beta allows the data to determine the trajectory of MA(t). The behavioral aspects of emotion are often couched in terms of a probabilistic impulse to action or action tendency. Accordingly, a key feature of the model is that behavior is probabilistically (not deterministically) linked to MA(t). If some particular behavior were a “true” indicator of MA in this model, its probability would increase linearly with MA throughout its entire functional range. The linkage function of this behavior would be a straight line with a positive slope. However, the preceding discussion suggests that there is no “true” indicator of MA and that different behaviors become most probable within different parts of the MA range. After examining a number of candidates for the linkage functions, including nonparametric approximations and negative exponentials, we chose a composite logit polynomial. The logit component handles binary variables appropriately; the polynomial terms provide the closest approximation to empirical observations while preserving the distinction between the linkage functions and the MA(t) term which is embedded within them (i.e., parameters of the linkage functions and of MA(t) can be estimated separately).

Estimation of model parameters required some complex and novel statistical manipulations because the observed behaviors are correlated (not independent), both at any particular moment in time and throughout the course of tantrum as well. For example, the occurrence of behavior X at time t_1 is not independent of the occurrence of behavior Y at t_1 or at any subsequent time. The steps and corresponding rationales in model estimation are outlined briefly as follows:

- (1) Tantrum durations were normalized to a 0–1.0 scale, thus permitting tantrums of different durations to be combined on the same scale.

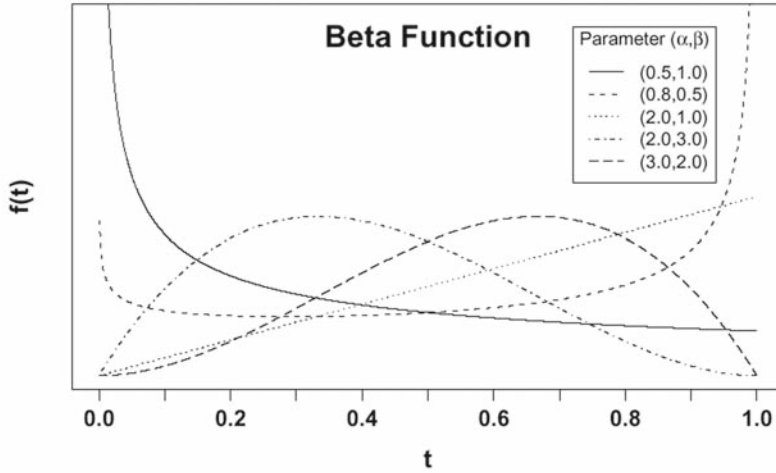


Fig. 12.1 Parametric variations in the shape of the beta function

- (2) The beta parameters a and b were reformulated as the exponential terms, $\exp(a)$ and $\exp(b)$. This reparameterization lifted the restriction that the parameters must remain positive (i.e., >0), thus permitting some of the required statistical manipulations. With these adjustments, $MA(t)$ assumes the following form:

$$MA(t, a, b) = t^{\exp(a)-1} (1-t)^{\exp(b)-1}, \text{ for } a, b \in (-\infty, \infty) \quad (1)$$

- (3) Initial modeling (Qiu, Yang, & Potegal, 2005) showed that the trajectory of $MA(t)$ shifted markedly with overall tantrum duration. In particular, the longer the tantrum, the more delayed was the peak of $MA(t)$. To accommodate such effects, the a and b parameters of $MA(t)$ are now formulated as a second-order polynomial of duration, d . Namely,

$$a = a_0 + a_1 d + a_2 d^2, b = b_0 + b_1 d + b_2 d^2 \quad (2)$$

where a_0, a_1, a_2, b_0, b_1 , and b_2 are unknown coefficients.

- (4) The probability of the k th angry behavior at each point throughout the normalized time of the tantrum is represented as $\pi_k(t)$ for $k = 1, 2, \dots, 8$. Because logit functions with linear terms alone were found insufficient to capture the complex relationships between MA and behavior likelihood, second-order polynomials were used to link $\pi_k(t)$ with $MA(t)$, yielding the linkage function equation

$$\log [\pi_k(t)/(1 - \pi_k(t))] = C_{0k} + C_{1k}MA(t, a, b) + C_{2k}MA(t, a, b)^2 \text{ for } k = 1, 2, \dots, 8 \quad (3)$$

- (5) The parameters, $a_0, a_1, a_2, b_0, b_1, b_2, c_{0k}, c_{1k}$, and c_{2k} , were then estimated using the well-accepted Generalized Estimating Equations technique (GEE, e.g., Liang & Zeger, 1986), which

is a generalization of least squares regression when the regression model is nonlinear. The GEE was chosen for this analysis rather than the commonly used alternative, the Newton–Raphson algorithm, because the latter requires specification of the likelihood function which is difficult to formulate explicitly for longitudinal data when the observations over time are nested (e.g., within subjects) and are all correlated, as they are here. By contrast, the GEE requires only the specification of the mean and variance functions, which are not difficult to formulate even when inter-variable correlations exist. Model-checking plots (Cook & Weisberg, 1999) were used as graphic goodness-of-fit heuristics in determining, e.g., the order of the polynomials for $MA(t)$ and linkage functions. Mathematically inclined readers are referred to Qiu, Yang, and Potegal (2009) for a fuller exposition.

12.3.4 Comparison of Wisconsin and Minneapolis Data

We used the eight behaviors that loaded on anger factors in both data sets. The model produced good fits to these data and met standard statistical criteria for stability. The iterative GEE algorithm converged to a solution reasonably rapidly and did so from a range of initial values (this was an improvement over results obtained with a simpler model of the WI data, Qiu et al., 2005). Notably, the MN data yielded a more stable solution than did the WI data. Because MA turns out to be a complex, joint function of time within the tantrum and overall tantrum duration, Fig. 12.2 compares $MA(t)$ for the MN (top row) and WI (bottom row) data as wire-frame time-duration surfaces. This figure shows the crucial result that the time-duration surfaces of the WI and MN data are reasonably similar. Their similarities include the following:

- (1) Within tantrums, $MA(t)$ typically rises steeply and falls more slowly. This is entirely in keeping with expectations about the time course of anger derived from self-reports (Potegal, this book). By definition, $MA(t) = 0$ at $t = 0$; the high initial value shown for $MA(t)$ on the graphs indicates that it rises to near-peak values by the end of the first time unit.
- (2) The longer the tantrum, the more delayed is its peak.
- (3) The peak height of anger varies as an inverted U-shaped function of tantrum duration. Tantrums lasting up to 9 min (in the WI data) or 15 min (in the MN data) show progressively higher peaks of MA. Tantrums which are longer than these respective durations (17% of the WI sample and 4% of the MN sample, respectively) have progressively lower peaks. There was good agreement in the coefficients of parameter a of the MA beta function. The surfaces do differ in the extent to which the peak shifts toward the end in the longer tantrums; the more rigorous procedures for collecting the MN data and the faster convergence in their modeling suggest that the surface generated from these data is a better estimate of population characteristics. Figure 12.3 provides simpler views of the trajectories of $MA(t)$ for tantrums that are 3, 6, 9, and 24 min long. Note the very rapid rise of $MA(t)$ in shorter tantrums. The rising phase of $MA(t)$ appears similar for the three shorter tantrums; the principal difference is in peak height. Thus, as a first approximation, the answer to the question raised in Section 12.2 is that the difference in most tantrums is not in the rate at which anger climbs, but in the peak value it achieves. In contrast to this plausible result, the finding that tantrums longer than 9–15 min are systematically less angry comes as a surprise (despite a previous analysis of the WI data which suggested a slower rise for the longer tantrums, Potegal, Kosorok, & Davidson, 1996). We are unaware of any published findings to this effect and the result may not accord with intuition. Why has nobody noticed? We suggest that it would be very difficult to retain estimates of moment-to-moment anger while observing a long tantrum. Instead, observers are more likely to form an impression of overall or cumulative

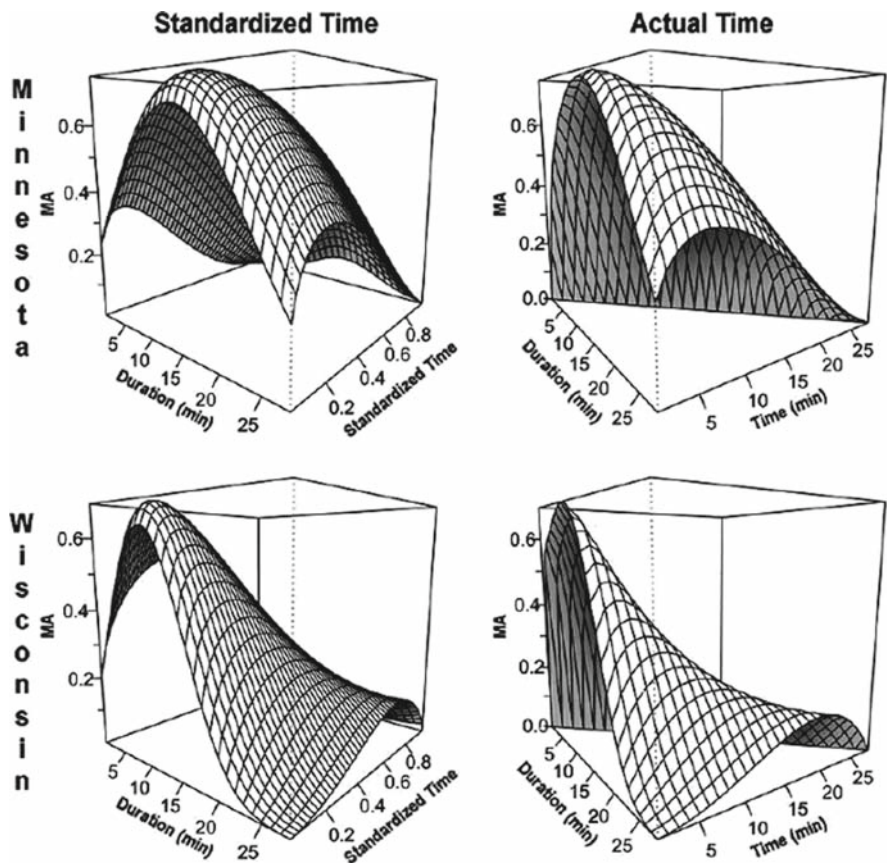


Fig. 12.2 Time within the tantrum is represented along the X-axis, overall tantrum duration is represented along the Y-axis, and $MA(t)$ is represented on the Z-axis. For each value of duration on the Y-axis, the corresponding X–Z curve displays the trajectory of $MA(t)$ over time for the idealized tantrum of that duration. The X-axis in the left column is the standardized time (0–1.0) used in our calculations. The same results are displayed in the right column, but with real time on the X-axis. The real time graphs depict the trajectory of $MA(t)$ and the location of its peaks more realistically, but are somewhat more difficult to read because of the brevity of the shorter tantrums

anger. Accordingly, the plots of cumulative $MA(t)$ over time in Fig. 12.4 show that “total anger” increases up to 15 min for the WI data and up to 20 min in the MN data. This implies that less than 8% of the WI tantrums or 2% of MN tantrums would have shown a reduced level of cumulative anger relative to shorter tantrums. That is, tantrums long enough to show reduced levels of anger are relatively rare.

The panels of Fig. 12.5 display pairwise comparisons of the linkage functions derived from the two data sets. The overall probabilities of each of the respective behaviors are in close agreement (i.e., the mean heights of the pair of functions for each behavior are similar). Furthermore, the functions for most of the behaviors have qualitatively similar shapes. Pairwise, all parameters agree in sign (e.g., a_0 for stiffen is negative in both WI and MN data), meaning that the parameters have similar effects on the linkage functions. Finally, there is overlap in the respective 95% confidence intervals for each of the three parameters for six of the eight behaviors. The WI and MN linkage functions for scream differ in one parameter. The major discrepancy is in the parameters for

Fig. 12.3 $MA(t)$ for the 3, 6, 9, and 24 min $X-Z$ duration planes of the real time (*right column*) plots of Fig. 12.2 are superimposed here on single graphs

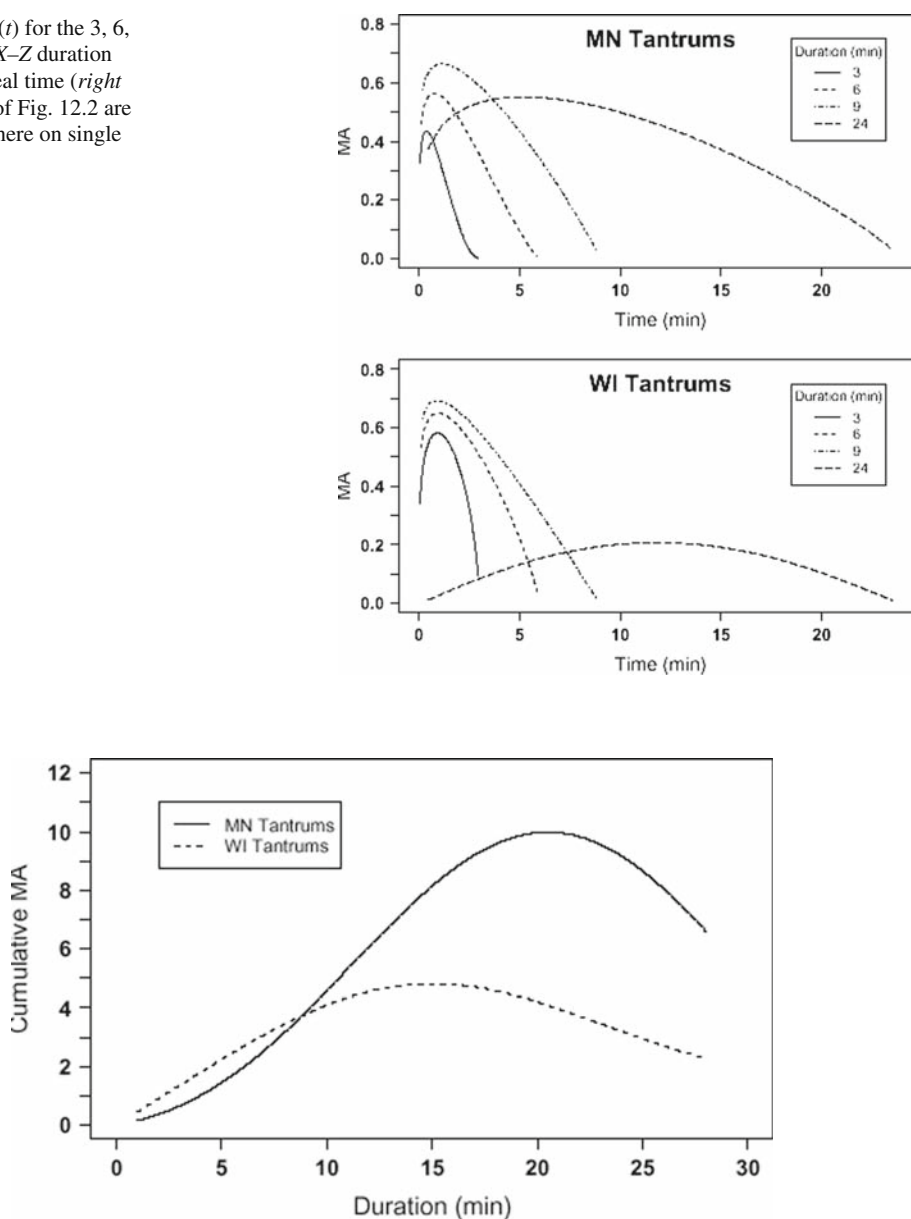


Fig. 12.4 Comparison of cumulative MA as a function of tantrum duration

arch/stiffen. Where results do differ, the parameters derived from the MN data are again better estimators of population characteristics. Although a linear term was included in all the linkage functions, as expected, none of them are strictly linear. That is, there is no “true” behavioral indicator of MA. Most functions show a maximum somewhere along the range of MA, indicating the level of anger at which that behavior is most likely. The lower anger behaviors, e.g., arch and throw, differ from the

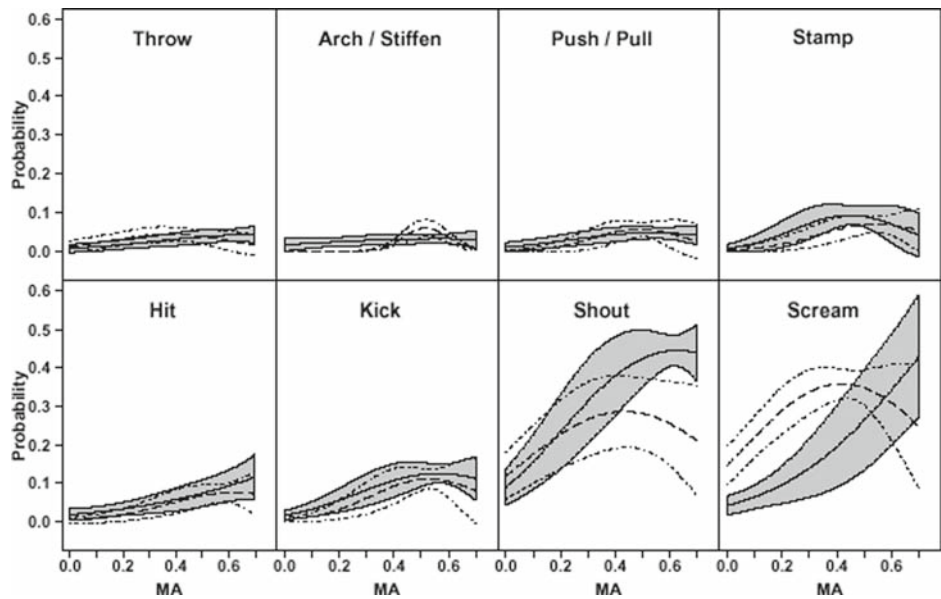


Fig. 12.5 Comparison of MN and WI linkage functions. *Dashed lines* are WI linkage functions ± 1 SD; *gray areas* are MN linkage functions ± 1 SD

higher anger behaviors, e.g., scream, shout, and kick, in that the probability of lower anger behaviors increases only slightly along the range of MA and they reach their respective maxima (i.e., they become most probable) at lower values of MA. In contrast, the probability of higher anger behaviors increases strongly with MA and they become most probable at higher values of MA. These effects are expressed by shallow, inverted U-shaped linkage functions for the lower anger behavior and steeper, right-shifted linkage functions for the higher anger behaviors. This is exactly the expected outcome, quantitatively capturing the qualitative relationship between internal anger intensity and corresponding external behavior that was the impetus for this model.

12.3.4.1 The MA50 as a Measure of the “Characteristic” Anger Intensity of Tantrum Behaviors

One major application is the estimation of anger intensity in particular events as expressed by the behaviors occurring in that event. Because the model calculations are complex, we offer a computational shortcut to anger estimation in the form of a set of anger “weights,” termed the MA50. The MA50 of an individual behavior is derived from its linkage function and reflects its ranking on the MA scale. Specifically, the MA50 for a behavior is defined as that MA value at which the probability of the behavior is midway between its initial and peak probabilities. The MA50 is analogous to the standard effective dose (ED50) and lethal dose (LD50) measures in pharmacology, which compare drug potencies by the dose necessary to produce a given effect in half the subjects. In Fig. 12.5, e.g., stamp in the MN data reaches its peak value (i.e., is most probable) at MA = 0.45; its MA50 = 0.23. In contrast, the probability of scream increases all the way to the end of the calculated MA range, MA = 0.7; the MA50 of scream is 0.47. Table 12.3 provides provisional MA50 values calculated from the linkage functions of the MN data. Because the convention in psychology is that weights

should be small, whole numbers, Table 12.3 also provides a list of suggested weights. These weights have been rounded up or down to increase consistency with the factor analytic results and to be conservative with regard to the magnitude of differences in characteristic intensity among behaviors. Overall anger intensity calculated with these weights can be correlated with other variables of interest (e.g., behavioral and/or autonomic arousal, judgments of the severity of anger, or risk of psychopathology) or used to differentiate among groups where differences in anger intensity are thought to be important, e.g., among children who are at low vs. high risk for externalizing disorder. Readers may wish to compare results obtained with unweighted vs. MA50 weighted scores or derive their own regression coefficients for the various behaviors and compare them to the listed values.

Table 12.3 “Characteristic” anger intensities of tantrum behaviors

Behavior	Provisional MA50	Suggested weight
Scream	0.47	4
Hit	0.47	4
Throw	0.32	3
Kick	0.26	3
Shout	0.25	3
Push	0.23	2
Stamp	0.23	2
Arch/stiffen	0.21	2

MA50 values are denoted as “provisional” because the current linkage functions, and the MA50 values calculated from them, are approximations to the “true” linkage functions (Section 3.2.2).

12.4 Current Experimental Limitations and Theoretical Assumptions

12.4.1 Methods

Data collection. The detailed analysis of the rising phase of $MA(t)$ is limited by our use of (1) the first occurrence of a tantrum behavior, rather than the tantrum triggering event, as the start point and (2) 30 s observational units. Both limitations were imposed by our observational techniques and are being rectified in ongoing studies with new methodology.

12.4.2 Modeling Assumptions, Functions, and Interpretations

The beta function. There is no a priori reason for the central nervous system to be in the business of generating beta functions. It was chosen for modeling $MA(t)$ because it can assume a variety of shapes, but it is only an approximation at best. We did obtain similar results with the lognormal and gamma functions. A particular limitation of the beta is that the rising and falling phases assume the sigmoid configuration typical of many biological growth functions only under a certain range of parameter values. However, modeling the rising phase of $MA(t)$ with a logistic function did not produce a better fit to the data.

Linkage functions. Because the rates at which various tantrum behaviors occur outside a tantrum are very low compared to their probability of occurrence within a tantrum, the initial values of the

linkage functions shown in Fig. 12.5 should all be 0. That they are not is a consequence of using the first behavior to occur as the start point of the tantrum.

Variability. The $MA(t)$ time-duration surfaces indicate that, up to some duration, the longer the tantrum, the higher is its MA peak. However, there is considerable variability in our data sets, which certainly include some tantrums that are short, but contain many angry behaviors.

Multiple peaks? Recall of emotion experiences by adults as well as some observations of children's tantrums suggests that one or more secondary peaks may be superimposed on the primary rapid rise and slower fall of anger (Frijda, Mesquita, Sonnemans, & Van Goozen, 1991 Fig. 7.2, Parens, 1993, Fig 7.1). Pooling and normalizing data to a 0–1.0 time axis was necessary to develop the current model, but precluded the possibility of detecting anger cycles with a brief period.

Other associations among angry behaviors. The alert reader will have noticed that similarities among linkage functions, and the ordering of MA50s derived from them, correspond only partially to the grouping of behaviors into intensity-related factors in the factor analyses. This is not necessarily a contradiction in that the factor analyses involve the cumulative durations of behaviors within tantrums while the linkage functions derive from the temporal distributions of behaviors. A likely explanation for the differences is that behaviors may load on the same factor for reasons other than a common drive by MA. For example, highly vocal children may shout and scream when $MA(t)$ is high, but not necessarily hit or kick. These same children may also be more likely to whine. This differentiation would be consistent with clustering of verbal vs. physical anger patterns in adults (Deffenbacher et al., 1996).

Alternative/future models of $MA(t)$. The attentive reader will have also noticed that time enters into our model directly in that MA is a straightforward function of time, but also indirectly in that the parameters of MA vary with overall tantrum duration. While the model provides a consistent fit to the data and new insight into the time course of anger, the effects of time would be more concisely represented in a single variable. Tantrums unfold over time depending upon initial conditions, the state and traits of the child, and on the interactions of these variables with events such as parental intervention. Overall duration in the current version of the model is presumably a stand-in for these collective effects. A future approach could involve dynamic equations, or numerical simulations, in which MA appears as an evolving function of time; time- and/or event-driven variation in the growth and decay parameters would capture the influence of relevant variables and processes. However, all these approaches require prior knowledge of the basic linkages between MA intensity and the behaviors that indicate anger. The current model provides this necessary knowledge.

$MA(t)$ vs. "anger." How does $MA(t)$ fit with the coupled response systems of anger? Our basic assumption, that anger scales along a single dimension of intensity, implies that significant variance in behaviors can be captured by a single variable, $MA(t)$, whose value represents overall anger intensity. This assumption also precludes the idea of multiple types of anger or distinctions between lower intensity anger and higher intensity rage suggested in several other chapters in this book. Note our care in using the term $MA(t)$, not anger, in our discussion. At one extreme, $MA(t)$ may be a classic latent variable, a statistical construct which functions as a convenient envelope for the set of behaviors observed and which reflects no deeper psychological or physiological process. At the other extreme, $MA(t)$ may describe the time course of measurable neural process(es) which control behavioral response tendencies as well as autonomic responses, subjective experience and their coupling. As such, calculated MA might have stronger correlations with, and higher predictive value for, e.g., experimentally measured autonomic arousal, self and other judged intensity of anger, impact on family life, level of childhood psychopathology than do any individual behavior or combination of behaviors. For the moment, we regard $MA(t)$ as a global variable that describes the envelope of the action tendencies and acts which comprise one subsystem of anger.

12.5 Some Future Extensions and Applications of the Model

12.5.1 *Facial, Vocal, and Autonomic Expression*

To this point, the model has addressed the probability of responses, but other response characteristics change in anger as well. The model should be extended to include increases in response “intensity” with anger (e.g., increments in amplitude and frequency of vocalizations and the speed, force, and repetition of physical acts). Even more importantly, the model should incorporate the traditional measures of facial, vocal, and autonomic anger expression. A strong form of the proposition that facial expressions are “gold standard” markers of emotions would be the claim that the linkage function for facial anger in our model would be linear over the full range of MA. Self-reported anger intensity is most closely related to the total number of co-occurring AUs (Alvarado & Jameson, 2002). However, although facial expressions of anger distinguished subjects in Stemmler’s (1997) study who reported having been slightly angered by a mild variant of an insult manipulation from those who had been exposed to the moderate or “full” anger variants, facial expressions did not distinguish the moderately from the “fully” angered subjects. These results suggest that angry expressions do not become progressively more pronounced as MA rises from baseline to its peak but saturate at some sub-maximal level. Stemmler (1997) did find that the slight, moderate, or “fully” angry groups showed correspondingly graded increases in diastolic blood pressure (DBP). The model would become more general, and more integrated across levels, by inclusion of linkage functions for these measures, too.

12.5.2 *Linkage Functions May Vary with Conditions*

The similarity between the linkage functions derived from the WI and MN data indicates their consistency across children. This does not mean that they are unchangeable. It is entirely plausible that linkage functions shift with stimulus conditions and/or experience. In the arm restraint study noted in Section 12.2, e.g., the finding that angry facial expression had the longest latency and lowest probability of the three responses suggests that its linkage function was displaced to the right. In this study, the child was seated on the parent’s lap facing away from her, i.e., there was no visible social stimulus for eliciting a facial expression. Functionalist accounts of “audience effects” on facial expression (cf. Hess, 2001) suggest that if the child were to see the parent’s face and visual communication were salient, facial expressions would become more probable. That is, the linkage function for facial anger might well shift up and/or left.

12.5.3 *Life Span Change vs. Continuity*

We assume continuity of anger dynamics across the life span in order to develop the implications of the model; we justify this assumption on the grounds that the two basic phenomena, a rise-and-fall trajectory and a differential association of various behaviors with low and high intensity anger, remain true in adulthood. The observations that tantrums increase in duration up to age 5, at which time they approximate the lower end of the range of adult episodes of anger, and are then reported not to change with age in adulthood (Potegal, this book) suggest that some $MA(t)$ parameters may remain fairly stable from middle childhood on. Episode-wise, self-reported intensity of anger increases with the degree of perceived injustice (Mikula, Scherer, & Athenstaedt, 1998) and relative social status (Edwards, 1998); these findings may reflect not only how $MA(t)$ is governed by social circumstances

but also how it is subjectively experienced by adults. Anger intensity is reported to drop across adulthood (Schieman, this book), suggesting a reduction in peak $MA(t)$ with age.

Linkage functions may be, perhaps, even more prone to change with development. Effective parental discipline for specific behaviors, such as biting or hitting, might shift the linkage functions for these behaviors down and/or to the right. In fact, the socialization of children away from the physical expression of anger might be nicely captured by corresponding overall shifts in linkage functions. On the other hand, some ordering of linkage functions may be preserved through the life span. Some victims of partner abuse report being shoved and slapped both *before* and *after* the main physical assault (Dobash & Dobash, 1984). This observation finds a natural explanation in that shoving and slapping are behaviors that occur at lower levels of MA, i.e., at the beginning of its rise and the end of its fall.

12.5.4 Sex Differences

Overall, many surveys indicate that the frequency and intensity of men's and women's experiences of anger do not differ (Fischer & Evers, this book). However, one notable sex difference is that women are much more likely to cry when angry (Vingerhoets & Scheirs, 2000). Reframing these findings in terms of the model, men and women may have similar $MA(t)$ curves, but their linkage function for crying differs markedly. Campbell and Muncer's (1994) finding that men typically see the expression of anger as seizing control of the situation and exerting dominance while women more typically view the expression of anger as a loss of self-control has been replicated in England (Archer & Haigh, 1999), Spain (Ramirez, Andreu, & Fujihara, 2001) and France (Richardson & Huguet, 2001). One current hypothesis is that women are more reluctant to express anger and do so only at higher intensities, which is when they are more likely to feel they have lost control (e.g., Astin, Redston, & Campbell, 2003). This hypothesis is supported by a meta-analysis showing greater likelihood of male aggression at low or moderate levels of "emotional arousal" (i.e., anger), but much less difference at high arousal levels (Knight, Guthries, Page, & Fabes, 2002). In the laboratory, women's longer latency to respond to continuing provocation has been accordingly interpreted as indicating their higher anger "flashpoint" for overt aggression (Zeichner, Parrott, & Frey, 2003). This interpretation has a natural and empirically testable explanation as a right shift of behavioral linkage functions for women.

12.6 Implications of the Model for Theory and Research on Anger

12.6.1 Appraisal

As argued by Potegal and Stemmler (this book), appraisal in some form is a necessary part of the neurological process of anger elicitation in adults. The same must be true of children. However, anger rises too quickly in the majority of tantrums for extensive appraisal to occur. Tantrums are well practiced and seemingly automatic. A child whose daily tantrums begin in earnest at 18 months will have had upwards of 500 tantrums by her third birthday. Such extensive practice probably contributes to tantrum automaticity and the rapid rise of $MA(t)$. In meeting the challenge of reconstructing the appraisal process in children's anger, Stein, Trabasso, and Liwag (1993), Fig. 20.2 developed a richly detailed flow diagram of cognitions and plans. As thoughtful and detailed as such diagrams were, they could not reflect events in real time because the requisite real-time data were not available.

Before conjecturing about how appraisal may drive children's anger, the trajectory of that anger, and the time frame into which any such processing must fit, should be established.

12.6.2 Display Rules: Anger Regulation in Social Context

Deliberate “minimization” and “maximization” of negative emotions can be seen by the second year of life (Saarni, 1993). These instances typically occur in a social context. The term “display rules” refers to socially learned rules that determine how much of felt emotion is shown, and to whom (e.g., Underwood, Coie, & Herbsman, 1992, Lemerise & Harper, this book). Restraining the expression of anger is more characteristic of girls than boys. As early as 4–5 years of age, girls receiving an unattractive gift express fewer negative emotions than do boys (e.g., Davis, 1995). In recalling their own experiences as well as in responding to vignettes, 7–12 year olds indicated that they modify their own emotion expression according to the age, sex, and social role of the person with whom they are interacting; girls report more anger suppression than boys (Underwood et al., 1992; Shipman, Zeman, Nesin, & Fitzgerald, 2003). Within the model, such display rules would naturally appear as a down shift or right shift of linkage functions. Thus, use of the linkage functions could help quantify these sex differences.

12.6.3 Caveats

The foregoing discussion suggests shifts or differences in $MA(t)$ and/or linkage functions as potential explanations for some normal and psychopathological anger-related phenomena. With this in mind, we note the need for care in applying the model. For example, MAs calculated separately for different groups will be comparable in certain respects, but not in others. Thus, peak locations can be directly compared. In contrast, although MA is a ratio measure (e.g., an MA of 0.4 is a level of intensity twice as great as a MA of 0.2), equivalent MA peak heights in different groups do not necessarily represent equal occurrences of angry behaviors. Specifically, if $MA(t)$ values are to be compared among groups, their data must be entered into the model together. At this early stage in model development, determining what may be happening in particular cases is likely to require rigorous modeling of an extensive data set collected for that specific purpose. Qualitative explanations of phenomena in terms of $MA(t)$ and/or linkage functions without such rigorous modeling may be more facile than true.

12.7 The Anger Intensity-Behavioral Linkage Function Model and the Brain

As described by Potegal and Stemmler (this book), meta-analyses of neuroimaging studies highlight the lateral orbitofrontal cortex (OFC) as an area very consistently activated in anger. Hemispheric asymmetries in EEG activation have consistently implicated left frontal cortex as a region that becomes activated with anger (e.g., Harmon-Jones, Sigelman, Bohlig, & Harmon-Jones, 2003). How does the anger intensity-behavioral linkage function model fit with, or facilitate the analysis of, the neural bases of anger? It is possible, of course, that $MA(t)$ is a classic latent variable, a statistical construct with no necessary physiological substrate. Alternatively, the beta may be methodologically useful in the curve fitting of neural activity. If, however, activation in lateral OFC and associated regions is the basis for an increased probability of anger-related behaviors, then $MA(t)$ may turn out to be the envelope of that activation. Our model implies that the order of appearance of

different angry behaviors is determined by their respective linkage functions, perhaps as successively higher thresholds along the MA axis. Here we note two precedents for how this process might be instantiated in the brain.

12.7.1 Spinal Motor Neurons

One physiological model for automatic escalation in the nervous system is the sequential recruitment of spinal motoneurons, in order of size, for a movement being executed, i.e., the well-established "size principle" (e.g., Cope & Pinter, 1995). Because the force that motor neurons elicit is an increasing monotonic function of their size, (i.e., the larger the unit, the more force it exerts), a consequence of the size principle is that force increases in a roughly linear fashion up to the requirements of the movement. The mechanism of the size principle is embedded in the neurophysiology of the spinal motor neuron pool. However, spinal circuitry operates in the millisecond range while the cerebral circuitry of anger functions in the second to minute range. Furthermore, because anger escalation involves cerebral rather than spinal circuitry, it may not function as rigidly as motor neuron recruitment. Thus, a more relevant model may be the neural control of fear responses.

12.7.2 The Temporal Organization of Anxiety and Fear

Fanselow (1994), Blanchard and Blanchard (2008), and others have shown that when an animal first detects a predator at a distance, it becomes immobile ("freezes"). If the predator comes closer it flees; if the predator closes in, the animal then counterattacks defensively. In McNaughton and Corr's (2004) generalized model of this hierarchy, an internalized perception of threat increases along a continuum (e.g., with increasing proximity of the predator); the behaviors in the hierarchy are triggered at successively higher levels of this continuum. These hierarchically triggered, but topographically distinct behaviors are generated by different neural circuits. Details of neuroanatomy and behavior may vary for species and individuals, but in exactly this sense, a recent neuroimaging study of humans being pursued by a "predator" in a virtual maze (but with real physical pain in the form of electric shock if the predator "catches" the avatar) demonstrated a very dramatic shift in the region of brain activation. Activation in orbitofrontal cortex and lateral amygdala associated with milder anxiety shifted to the periaqueductal gray and central amygdala as the predator closed in and fear intensified (Mobbs et al., 2007). We suggest that response shift with increasing MA(*t*) may similarly involve sequential recruitment of different neuroanatomical systems in the brain.

Explosive growth in affective neuroscience is just beginning and the sophisticated physiological measures in use should be complemented by the most objective, behaviorally based measures of emotion available. We hope that the ideas for quantifying anger presented here will facilitate that growth.

Acknowledgments Collection of the Wisconsin data was supported by a grant to M. Potegal from the Harry Frank Guggenheim Foundation and by National Research Service Awards to M. Potegal from the National Institute for Neurological Disorders and Stroke (F33 NS09638) and the National Institute of Child Health and Human Development (F33 HD08208). At that time, the first author was a Fellow in the laboratory of Richard J. Davidson, where work was supported in part by an NIMH Center for Behavioral Sciences Research Grant (P50-MH52354) to the Wisconsin Center for Affective Neuroscience (R.J. Davidson, Director) and by an NIMH Research Scientist Award (KO5-MH00875). Collection of the Minnesota data and development of the anger intensity-linkage function model were supported by grants to M. Potegal from the National Institute for Mental Health (R03-MH58739) and from the National Institute of Child Health and Human Development (R21 HD048426); this second stage was also supported

by a Grant-in-Aid from University of Minnesota Graduate Faculties (Emotion and manipulation in toddler tantrums: Behavior/cortisol relations), the Viking Children's Fund (643-7257) and an equipment grant from the Minnesota Medical Foundation.

We thank the University of Minnesota undergraduates who coded the various Minnesota tantrum records including Vanessa Downs, Ross Oden, and Jennifer B. Swanson. We thank Kristin Buss, Nancy Eisenberg, James Green, Jan Hoeksma, Jill Kilderman, Alicia Kunin-Batson, Harriet Oster, and Cynthia Stifter for their critical reading of earlier versions of this paper, in part or in whole. David Mottet improved the graphics of Fig. 12.2.

References

- Alvarado, N., & Jameson, K. A. (2002). Varieties of anger: The relation between emotion terms and components of anger expression. *Motivation and Emotion*, 26, 153-182.
- Archer, J., & Haigh, A. (1999). Sex differences in beliefs about aggression: Opponent's sex and the form of aggression. *British Journal of Social Psychology*, 38, 71-84.
- Astin, S., Redston, P., & Campbell, A. (2003). Sex differences in social representations of aggression: Men justify, women excuse? *Aggressive Behavior*, 29, 128-133.
- Blanchard, D. C., & Blanchard, R. J. (2008). Defensive behaviors, fear, and anxiety. In R. J. Blanchard, D. C. Blanchard, G. Greibel, & D. J. Nutt (Eds.), *Handbook of anxiety and fear* (pp. 63-80). Amsterdam: Elsevier.
- Bonano, G. A., & Keltner, D. (2004). The coherence of emotion systems: Comparing "on-line" measures of appraisal, facial expressions, and self-report. *Cognition and Emotion*, 18, 431-444.
- Campbell, A., & Muncer, S. (1994). Sex differences in aggression: social representation and social roles. *British Journal of Social Psychology*, 33, 233-240.
- Camras, L., Sullivan, J., & Michel, G. (1993). Do infants express discrete emotions? Adult judgments of facial, vocal and body actions. *Journal of Nonverbal Behavior*, 17, 171-186.
- Casey, R. (1993). Children's emotional experience: Relations among expression, self-report, and understanding. *Developmental Psychology*, 29, 119-129.
- Coan, J. A., & Allen, J. B. (2003). Varieties of emotional experience during voluntary emotional facial expressions. *Annals of the New York Academy of Science*, 1000, 375-379.
- Cole, P. M., Teti, L. O., & Zahn-Waxler, C. (2003). Mutual emotion regulation and the stability of conduct problems between preschool and early school age. *Development and Psychopathology*, 15, 1-18.
- Cook, R. D., & Weisberg, S. (1999). *Applied regression including computing and graphics*. New York: Wiley.
- Cope, T. C., & Pinter, M. J. (1995). The size principal of motoneuron recruitment: Still working after all these years. *News in Physiological Sciences*, 10, 280-286.
- Davidson, R. J. (1992). Prolegomenon to the structure of emotion: Gleanings from neuropsychology. *Cognition and Emotion*, 6, 245-268.
- Davis, T. L. (1995). Gender differences in masking negative emotions: Ability or motivation? *Developmental Psychology*, 31, 660-667.
- Dearing, K. F., Hubbard, J. A., Ramsden, S. R., Parker, E. H., Relyea, N., Smithmyer, C. M., et al. (2002). Children's self-reports about anger regulation: Direct and indirect links to social preference and aggression. *Merrill-Palmer Quarterly*, 48, 308-335.
- Deffenbacher, J. L., Oetting, E. R., Lynch, R. S., & Morris, C. (1996). The expression of anger and its consequences. *Behavior Research and Therapy*, 34, 575-590.
- Denham, S., & Couchoud, E. (1990). Young preschoolers understanding of emotions. *Child Study Journal*, 20, 171-191.
- Dobash, R. E., & Dobash, R. P. (1984). The nature and antecedents of violent events. *British Journal of Criminology*, 24, 269-288.
- Eckhardt, C. I., & Deffenbacher, J. L. (1995). Diagnosis of anger disorders. In H. Kassinove (Eds.), *Anger disorders: Definition, diagnosis and treatment* (pp. 27-48). Washington, DC: Taylor & Francis.
- Edwards, P. (1998). *Etude empirique de déterminants de la différenciation des émotions et de leur intensité*. [An empirical study of the determinants of the differentiation and the intensity of the emotions.] Ph.D. Thesis. University of Geneva, Switzerland.
- Einon, D. F., & Potegal, M. (1994). Temper tantrums in young children. In M. Potegal & J. Knutson (Eds.), *Dynamics of aggression: Biological and social processes in dyads and groups* (pp. 157-194). Hillsdale, NJ: Erlbaum Assoc.
- Eisenberg, N., Fabes, R. A., Murphy, B. C., Shaeperd, S., Guthrie, I. K., Maszk, P., et al. (1999). Prediction of elementary school children's socially appropriate and problem behavior from anger reactions at 4-6 years. *Journal of Applied Developmental Psychology*, 20, 119-142.

- Ekman, P. (1994). Strong evidence for universals in facial expressions: A reply to Russell's mistaken critique. *Psychological Bulletin*, 115, 268–287.
- Fanselow, M. S. (1994). Neural organization of the defensive behavior system responsible for fear. *Psychonomic Bulletin & Review*, 1, 429–438.
- Fernandez-Dols, J.-M., Sanchez, F., Carrera, P., & Ruiz-Belda, M.-A. (1997). Are spontaneous expressions and emotions linked? An experimental test of coherence. *Journal of Nonverbal Behavior*, 21, 163–177.
- Feshbach, S. (1964). The function of aggression and the regulation of aggressive drive. *Psychological Review*, 71, 257–272.
- Freud, A. (1971). Comments on aggression. *International Journal of Psychoanalysis*, 53, 163–171.
- Fridlund, A. J. (1997). The new ethology of human facial expressions. In J. A. Russell & J. M. Fernandez-Dols (Eds.), *The psychology of facial expression* (pp. 103–129). Cambridge University Press.
- Frijda, N. H., Mesquita, B., Sonnemans, J., & Van Goozen, S. (1991). The duration of affective phenomena or emotions, sentiments, and passions. In K. T. Strongman (Eds.), *International review of studies of emotion* (Vol. 1, pp. 187–226). New York: Wiley & Sons.
- Frijda, N. H., Ortony, A., Sonnemans, J., & Clore, G. (1992). The complexity of intensity. In M. Clark et al. (Eds.), *Emotion review of personality and social psychology* (pp. 60–89). 13 Newbury Park, CA: Sage Publications.
- Gross, J. J. (1998). The emerging field of emotion regulation: An integrative review. *Review of General Psychology*, 2, 271–299.
- Haidt, J., & Keltner, D. (1999). Culture and facial expression: Open-ended methods find more expressions and a gradient of recognition. *Cognition and Emotion*, 13, 225–266.
- Harmon-Jones, E., Sigelman, J. D., Bohlig, A., & Harmon-Jones, C. (2003). Anger, coping, and frontal cortical activity: The effect of coping potential on anger-induced left frontal activity. *Cognition and Emotion*, 17, 1–24.
- Hess, U. (2001). The communication of emotion. In A. Kaszniak (Ed.), *Emotions, qualia and consciousness* (pp. 397–409). Singapore: World Scientific Publishing.
- Hess, U., Blairy, S., & Kleck, R. (1997). The intensity of emotional facial expressions and decoding accuracy. *Journal of Nonverbal Behavior*, 21, 241–257.
- Hoeksma, J. B., Oosterlaan, J., & Schipper, E. (2004). Emotion regulation and the dynamics of feelings: A conceptual and methodological framework. *Child Development*, 75, 354–360.
- Hubbard, J. A., Parker, E. H., Ramsden, S. R., Flanagan, K. D., Nicole Relyea, N., Dearing, K. F., et al. (2004). The relations among observational, physiological, and self-report measures of children's anger. *Social Development*, 13, 14–39.
- Huesmann, L. R., Eron, L. D., Lefkowitz, M. M., & Walder, L. O. (1984). Stability of aggression over time and generations. *Developmental Psychology*, 20, 1120–1134.
- Knight, G. P., Guthrie, I. K., Page, M. C., & Fabes, R. A. (2002). Emotional arousal and gender differences in aggression: A meta-analysis. *Aggressive Behavior*, 28, 366–393.
- Kuppens, P., Mechelen, I., & Van Meulders, M. (2004). Every cloud has a silver lining: interpersonal and individual differences determinants of anger-related behaviors. *Personality & Social Psychology Bulletin*, 30, 1550–1564.
- Leibenluft, E., Charney, D. S., Towbin, K. E., Bhangoo, R. K., & Pine, D. S. (2003). Defining clinical phenotypes of juvenile mania. *American Journal of Psychiatry*, 160, 430–437.
- Levine, L., Stein, N. L., & Liwag, M. D. (1999). Remembering children's emotions: Sources of concordant and discordant accounts between parents and children. *Developmental Psychology*, 35, 790–801.
- Liang, K. Y., & Zeger, S. L. (1986). Longitudinal analysis using generalized linear models. *Biometrika*, 73, 13–22.
- Mascolo, M. F., Harkins, D., & Harakal, T. (2000). The dynamic construction of emotion: Varieties of anger. In M. D. Lewis & I. Granic (Eds.), *Emotion, development & self-organization* (pp. 125–154). Cambridge: Cambridge University Press.
- Matsumoto, D. (1989). Face, culture, and judgements of anger and fear: Do the eyes have it? *Journal of Nonverbal Behavior*, 13, 171–188.
- Matsumoto, D., Nezlek, B., & Koopmann, B. (2007). Evidence for universality in phenomenological emotion response system coherence. *Emotion*, 7, 57–67.
- McNaughton, N., & Corr, P. J. (2004). A two-dimensional neuropsychology of defense: Fear/anxiety and defensive distance. *Neuroscience & Biobehavioral Reviews*, 28, 285–305.
- Mikula, G., Scherer, K. R., & Athenstaedt, U. (1998). The role of injustice in the elicitation of differential emotional reactions. *Journal of Personality and Social Psychology*, 74, 769–783.
- Mirowsky, J., & Ross, C. E. (1995). Sex differences in distress: Real or artifact? *American Sociological Review*, 60, 449–468.

- Mobbs, D., Petrovic, P., Marchant, J. L., Hassabis, D., Nikolaus Weiskopf, N., Seymour, B., et al. (2007). When fear is near: Threat imminence elicits prefrontal-periaqueductal gray shifts in humans. *Science*, 317, 1079–1083.
- Nagin, D. S., & Tremblay, R. E. (2001). Parental and early childhood predictors of persistent physical aggression in boys from kindergarten to high school. *Archives of General Psychiatry*, 58, 389–394.
- Orbido de Castro, B., Merk, W., Koops, W., Veerma, J. W., & Bosch, J. D. (2005). Emotions in social information processing and their relations with reactive and proactive aggression in referred aggressive boys. *Journal of Clinical Child and Adolescent Psychology*, 34, 105–116.
- Parens, H. (1993). Rage toward the self and others in early childhood. In R. A. Glick & S. P. Roose (Eds.), *Rage, power, and aggression. The role of affect in motivation, development, and adaptation* (Vol. 2, pp. 123–147). New Haven, CT: Yale University Press.
- Plutchik, R. (1980). *Emotion: A psychoevolutionary synthesis*. New York: Harper & Row.
- Potegal, M. (2003, April). *Preliminary observations on salivary cortisol baseline and transients associated with tantrums in 3 year olds*. Paper presented at the biennial meeting of the Society for Research in Child Development Society for Research in Child Development, Tampa, FL.
- Potegal, M. (2005, April). *Tantrums in externalizing, internalizing and typically developing 4 year olds*. Poster presented at the biennial meeting of the Society for Research in Child Development: Atlanta, GA.
- Potegal, M., Robison, S., Anderson, F., Jordan, C., & Shapiro, E. (2007). Sequence and priming in 15 month olds' reactions to brief arm restraint: Evidence for a hierarchy of anger responses. *Aggressive Behavior*, 33, 1–11.
- Potegal, M., & Archer, J. (2004). Sex differences in childhood anger and aggression. *Child and Adolescent Psychiatric Clinics of North America: Sex and Gender*, 13, 513–528.
- Potegal, M., Carlson, G., Margulies, D., Gutkovitch, Z., & Wall, M. (2009). Rages or temper tantrums? The behavioral organization, temporal characteristics, and clinical significance of angry-agitated outbursts in child psychiatry inpatients. *Child Psychiatry and Human Development*, 40, 621–636.
- Potegal, M., & Davidson, R. J. (2003). Temper tantrums in young children: (1) Behavioral composition. *Journal of Developmental and Behavioral Pediatrics*, 24, 140–147.
- Potegal, M., Kosorok, M. R., & Davidson, R. J. (1996). The time course of angry behavior in the temper tantrums of young children. *Annals of the New York Academy of Sciences*, 794, 31–45.
- Potegal, M., Kosorok, M. R., & Davidson, R. (2003). Temper tantrums in young children: (2) Tantrum duration and temporal organization. *Journal of Developmental and Behavioral Pediatrics*, 24, 148–154.
- Qiu, P., Yang, R., & Potegal, M. (2005). *Generalized nonlinear modeling of the time course of tantrum anger*. Technical Report, School of Statistics, University of Minnesota, Minneapolis, MN 55455.
- Qiu, P., Yang, R., & Potegal, M. (2009). Statistical modeling of the time course of tantrum anger *Annals of Applied Statistics*, 3, 1013–1034.
- Radke-Yarrow, M., & Kochanska, G. (1990). Anger in young children. In N. L. Stein, B. Levinthal, & T. Trabasso (Eds.), *Psychological and biological approaches to emotion* (pp. 297–310). Hillsdale, NJ: Lawrence Erlbaum.
- Ramirez, J. M., Andreu, J. M., & Fujihara, T. (2001). Cultural and sex differences in aggression: A comparison between Japanese and Spanish students using two different inventories. *Aggressive Behavior*, 27, 313–322.
- Richardson, D. S., & Huguet, P. (2001). Beliefs about and experience with aggression in the United States and France. In J. M. Ramirez & D. S. Richardson (Eds.), *Cross-cultural approaches to aggression and reconciliation* (pp. 73–85). New York: Nova Science.
- Roseman, I. J., Wiest, C., & Swartz, T. S. (1994). Phenomenology, behaviors and goals differentiate discrete emotions. *Journal of Personality and Social Psychology*, 67, 206–221.
- Rosenberg, E., & Ekman, P. (1994). Coherence between expressive and experiential systems in emotion. *Cognition and Emotion*, 8, 201–229.
- Ross, C. E., & Van Willigen, M. (1996). Gender, parenthood, and anger. *Journal of Marriage and the Family*, 58, 572–584.
- Russell, J. A. (1995). Facial expressions of emotion: What lies beyond minimal universality? *Psychological Bulletin*, 118, 379–391.
- Russell, J. A. (1997). Reading emotions from and into faces: Resurrecting a dimensional-contextual perspective. In J. Russell & J. M. Fernandez-Dols (Eds.), *The psychology of facial expression* (pp. 295–320). Cambridge: Cambridge University Press.
- Saarni, C. (1993). Socialization of emotion. In M. Lewis & J. Haviland (Eds.), *Handbook of emotion* (pp. 435–446). New York: Guilford Press.
- Shaver, P., Wu, S., & Schwartz, J. C. (1992). Cross-cultural similarities and differences in emotion and its representation: A prototype approach. In M. Clark (Ed.), *Review of personality and social psychology Vol 13: Emotion* (pp. 175–212). Thousand Oaks, CA: Sage.

- Shipman, K. L., Zeman, J., Neshin, A. E., & Fitzgerald, M. (2003). Children's strategies for displaying anger and sadness: What works with whom? *Merrill-Palmer Quarterly*, 49, 100–122.
- Snyder, J., Stoolmiller, M., Wilson, M., & Yamamoto, M. (2003). Child anger regulation, parental responses to children's anger displays, and early child antisocial behavior. *Social Development*, 12, 335–360.
- Sonnemans, J., & Frijda, N. H. (1994). The structure of subjective emotional intensity. *Cognition and Emotion*, 4, 329–350.
- Stein, N. L., Trabasso, T., & Liwag, M. (1993). The representation and organization of emotional experience: Unfolding the emotion episode. In M. Lewis & J. M. Haviland (Eds.), *Handbook of emotions* (pp. 279–300). New York: Guilford Press.
- Stemmler, G. (1997). Selective activation of traits: Boundary conditions for the activation of anger. *Personality and Individual Differences*, 22, 213–233.
- Tassinanry, L. G., & Cacioppo, J. T. (1992). Unobservable facial actions and emotion. *Psychological Science*, 3, 28–33.
- Underwood, M. K., & Bjornstad, G. B. (2001). Children's emotional experience of peer provocation: The relation between observed behaviour and self-reports of emotions, expressions, and social goals. *International Journal of Behavioral Development*, 25, 320–330.
- Underwood, M. K., Coie, J. D., & Herbsman, C. R. (1992). Display rules for anger and aggression in school-age children. *Child Development*, 63, 366–380.
- Vingerhoets, A., & Scheirs, J. (2000). Sex differences in crying: Empirical findings and possible explanations. In A. H. Fischer (Ed.), *Gender and emotion* (pp. 143–165). Cambridge: Cambridge University Press.
- Wallbott, H. (1998). Bodily expression of emotion. *European Journal of Social Psychology*, 28, 879–896.
- Winstok, Z. (2007). Perceptions, emotions, and behavioral decisions in conflicts that escalate to violence. *Motivation and Emotion*, 31, 125–136.
- Zeichner, A., Parrott, D. J., & Frey, F. C. (2003). Gender differences in laboratory aggression under response choice conditions. *Aggressive Behavior*, 29, 95–106.

Chapter 13

The Development of Anger from Preschool to Middle Childhood: Expressing, Understanding, and Regulating Anger

Elizabeth A. Lemerise and Bridgette D. Harper

Abstract A review is provided of the developmental course of anger during the preschool years (3–5 years) and middle childhood (6–12 years). In addition, individual differences in anger during these developmental periods are reviewed. Three main aspects of anger are the focus of this chapter: (a) expression of anger, (b) perception and understanding of anger, and (c) regulation of anger. From the preschool years through middle childhood and beyond, children make great strides in the perception and understanding of anger and in regulating the anger they feel and express. These developmental changes are supported by advances in children's perceptual and cognitive development and by sensitive and responsive caregiving. The preschool years are a key time in the socialization of anger as perceptual, cognitive, and language development provide important tools for identifying, understanding, and regulating anger. Converging evidence demonstrates that, by the time of school entry, those children who have not mastered these skills are at risk for peer relations problems, poor adjustment to school, and a variety of externalizing problems.

In this chapter, we trace the developmental course of and individual differences in three aspects of anger: (a) expression of anger, (b) perception and understanding of anger, and (c) regulation of anger. Our focus is on the preschool period (3–5 years) and middle childhood (6 to 11–12 years), but some reference will be made to both earlier and later periods of development. We argue that all three aspects of anger develop in the context of interpersonal interactions in transactions with the social environment. In addition, perceptual/cognitive development and individual differences in temperament are essential to understanding the developmental course of anger in childhood.

13.1 Expressing Anger: Learning When, to Whom, and How

Based on experimental laboratory paradigms, observational studies, and maternal report, children's expression of anger has been shown to be associated with goal blockage (e.g., Buss & Goldsmith, 1998; Carpenter & Halberstadt, 2000; Goodenough, 1931; Lewis & Ramsay, 2005). Accordingly, and consistent with the functionalist perspective on emotion, anger function is to overcome obstacles in order to achieve goals (Saarni, Campos, Camras, & Withington, 2006). Despite its adaptive value, anger repels others and is associated with a variety of risks, ranging from children's externalizing

E.A. Lemerise (✉)

Department of Psychology, Western Kentucky University, Bowling Green, KY 42101-1030, USA
e-mail: elizabeth.lemerise@wku.edu

problems, peer rejection, and victimization (Casey & Schlosser, 1994; Cole, Teti, & Zahn-Waxler, 2003; Eisenberg, Fabes, Nyman, Bernzweig, & Pineulas, 1994) to adult susceptibility to heart disease (e.g., Barefoot, Dodge, Peterson, Dahlstrom, & Williams, 1989). This makes the regulation and culturally appropriate expression of anger a key developmental task. Thus individuals must learn “display rules” about when, to whom, and how to express emotions in culturally acceptable ways. These display rules for anger can be quite variable from culture to culture, leading to quite different parental reactions to anger displays in young children (see, e.g., Cole, Tamang, & Shrestha, 2006; Chapter 8).

Indirect socialization of display rules can be observed quite early in infancy wherein parents respond differentially to infants’ emotions (Malatesta & Haviland, 1982); this differential reinforcement is associated with declines in the expression of anger (and other negatively valenced emotions) and increases in neutral and positive expressions of emotion over the course of the first year of life. Declines in the expression of anger from 14 to 33 months also are related to secure attachment; whereas children who have insecure attachment relationships with caregivers express higher levels of negative emotions, including anger (Kochanska, 2001). Note that in the many samples of children and parents in whom attachment has been studied worldwide, secure attachment is the most common outcome across samples (van Ijzendoorn & Sagi, 1999). Thus, for most children, sensitive and responsive care from parents and other caregivers is related to reductions in the expression of anger and other negative emotions. However, the insensitive, unresponsive, and/or inconsistent caregiving received by some children is associated with higher levels of negative emotions, including anger. Presumably, such caregiving may create frustrations and goal blockages for children, leading to more anger and other negative emotions (see also Lewis & Ramsay, 2005).

Indirect socialization continues as children get older (e.g., Denham, 1993), but with children’s increasing age, parents use more direct socialization techniques as well (Denham, 1998; Lemerise & Dodge, 2008). Parental socialization of anger (and other emotions) is complicated by individual differences in temperament. In particular, researchers have found that some toddlers are “anger-prone” in that they are easily frustrated in laboratory tasks and are reported by their mothers to express more anger and to be less soothable, compared to other children (Calkins, Dedman, Gill, Lomax, & Johnson, 2002; Kochanska, Aksan, & Carlson, 2005). For these anger-prone children, supportive caregiving that reduces stress and fosters emotional competence is especially crucial (Blair, 2002; Kochanska et al., 2005). In particular, Blair (2002) has argued that this supportive caregiving fosters the regulation of strong emotions, supporting the development of effortful control and executive functioning. Conversely, when parents and children are both anger-prone (possibly due to a shared genetic susceptibility to difficult temperament, see e.g., Reuter, this volume), parents’ anger displays are likely to exacerbate children’s anger and dysregulation. When children are overwhelmed by strong emotions like anger, they may attempt to regulate these emotions by withdrawing from situations or by acting out. Both of these strategies are not only ineffective but also make it less likely that children will experience the kind of supportive interactions with adults and peers that foster social and emotional competence.

Compared to infants and toddlers, preschool-age children express anger less frequently, but they still have relatively poor control over their displays of anger and other emotions (Denham, 1998). Preschoolers not only need to coordinate goals with caregivers but must also, increasingly, coordinate goals with peers. Indeed, it has been argued that a key developmental task for preschool-age children is to learn to manage emotional arousal in order to engage in play with peers (Parker & Gottman, 1989). Anger can be readily observed among preschoolers in peer play contexts (e.g., Arsenio & Killen, 1996; Eisenberg et al., 1994), and research shows that individual differences in anger displays are related to social competence with peers such that children who have better control of anger are better liked by peers, are seen as more socially competent by teachers, and have lower risks for later

problem behaviors (e.g., Arsenio, Cooperman, & Lover, 2000; Eisenberg, et al., 1996; 1997; 2005; Fabes, Hanish, Martin, & Eisenberg, 2002).

These individual differences in preschoolers' expression of anger most likely are the result of a transaction between individual differences in temperamentally based anger-proneness and socialization of anger within the family context. Preschoolers' mastery of language provides a powerful tool for the socialization of emotion in that parents can directly socialize children's emotions through coaching or teaching, although parents vary in how effectively they coach emotions (Denham, 1998; Gottman, Katz, & Hooven, 1997). More effective socialization involves acknowledging and labeling the emotion, combined with coaching on constructive strategies for coping. Less effective parental socialization of emotions involves denial or minimization of the emotion and no coaching; children of these parents display poorer emotion knowledge and emotion regulation.

Parents also socialize anger indirectly in preschoolers and older children by providing models of expressivity (are children protected versus exposed to conflict and arguments, for example) and opportunities to learn about emotion via regulation of access to peers, stimulating games, and media outlets (Eisenberg, Cumberland, & Spinrad, 1998; Parke, 1994). Converging evidence shows that exposure to negative emotions (especially intense, reciprocated, and more frequent anger) in the context of parent-child interaction is associated with poor outcomes for children, including poorly regulated anger, aggression, and externalizing disorders (e.g., Caspi, et al., 2004; Cole et al., 2003; Denham, Mitchell-Copeland, Strandberg, Auerbach, & Blair, 1997; Hayden, Klein, & Durbin, 2005; Rubin, Burgess, Dwyer, & Hastings, 2003; Smeeckens, Riksen-Walraven, & van Bakel, 2007; Snyder, Stoolmiller, Wilson, & Yamamoto, 2003; Snyder, Schrepferman, McEachern, & Deleeuw, this volume). Exposure to inter-adult anger has similar outcomes, with effects being stronger for children who are higher in negative emotionality (e.g., Davies & Cummings, 1995; Jenkins, 2000; Jenkins, Shapka, & Sorenson, 2006; Schudlich, Shamir, & Cummings, 2004).

Another way in which parents indirectly socialize preschoolers' display rules for anger is through their own affective expressions. For example, parents' use of wrinkled brow and lip-biting strategies to minimize angry emotions predicted preschoolers' use of these same display rules to minimize their displays of anger both concurrently and longitudinally (Malatesta, Culver, Tesman, & Shepard, 1989). By the age of 4, children are aware of when and where to use display rules for anger and can successfully modify their expressions based on the current context. For example, Zeman, Penza, Shipman, and Young (1997) demonstrated that 4-year-olds were more likely to regulate their displays of anger with friends and with their mothers than with their fathers. Children reported that they believed that negative emotions, including anger, expressed with parents would lead to support or assistance, whereas friends would be less receptive to angry emotions (Zeman et al., 1997). Preschoolers' anger is also socialized indirectly within the daily context of their home environments. Preschool-age children reared in more negative, hostile environments are much less likely to use display rules to mask negative emotions, including anger, and are more likely to demonstrate mutual anger with mothers and to exhibit conduct problems in school (Cole et al., 2003). However, mothers who created predominantly positive and warm environments had preschoolers who displayed enhanced ability to display appropriate emotions, even in disappointing situations (Cole et al., 2003; Cole, Zahn-Waxler, & Smith, 1994).

For school-age children, the peer context becomes especially important for socializing anger due to children's sensitivity to their status in the peer group and strong peer group norms for regulating anger (Lemerise & Dodge, 2008; Parker & Gottman, 1989). By school age, difficulties in regulating anger are associated with rejection and victimization by peers (e.g., Eisenberg, Fabes, Guthrie, & Reiser, 2000; Hanish, et al., 2004). However, the ability to mask negative emotions and the display of positive emotions around peers is predictive of peer acceptance (Hubbard & Coie, 1994; Vosk, Forehand, & Figueroa, 1983). School-age children seem to understand this as they report masking

anger more than other emotions around their peers (Underwood, 1997) and using display rules more often with peers and less often with their parents (Zeman & Shipman, 1998). Children in middle childhood also understand that negative emotional expression, particularly anger expressed through aggression or sulking, is frowned upon by peers (Shipman, Zeman, Nesin, & Fitzgerald, 2003), but they also have lower self-efficacy for regulating anger than sadness (Zeman & Shipman, 1997). Children, regardless of peer group status, demonstrate accurate knowledge of when and where anger should be expressed (Underwood, 1997); however, not all children are successful in acting on this knowledge.

13.2 Perceiving and Understanding Anger

Across the preschool years, middle childhood, and adolescence, children's emotion perception accuracy improves; generally, accuracy is better for higher intensity stimuli and older children are better at some emotions than younger children (especially fear and disgust). Age differences tend to be greater when the emotion perception task requires verbal ability (e.g., labeling versus matching, see Denham, 1998; Herba, Landau, Russell, Ecker, & Phillips, 2006). Emotion perception is less accurate for some clinical groups (e.g., Blair, Colledge, Murray, & Mitchell, 2001; Castelli, 2005), and there is a growing body of research that suggests anger perception bias and/or poor emotion understanding is related to risk status. For example, Pollak and Tolley-Schell (2003) reported that physically abused children (8–11 years) displayed heightened attention to angry faces, with this heightened attention resulting in an advantage on tasks where the anger cue was relevant and a disadvantage when the cue was not valid. Other research has shown that in the context of a lab task where children and mothers discuss times the child felt happy, sad, and angry, maltreating mothers were less likely to engage in discussion reflective of emotion understanding, and their children scored lower on measures of emotion understanding than did matched nonmaltreated controls (Shipman & Zeman, 1999). Anger perception bias in 3–6-year-old children is also predicted by hostile maternal appraisals during a story reading session conducted 1 year earlier (Root & Jenkins, 2005) and by harsh parenting (Fine, Trentacosta, Izard, Mostow, & Campbell, 2004). Finally, a bias toward perceiving anger in others has been linked with later risks for aggression in both high-risk and low-risk populations of children (Fine et al., 2004; Schultz, Izard, & Bear, 2004; Schultz & Shaw, 2003).

The higher risks for aggression associated with anger perception bias can be explained via social information processing (Crick & Dodge, 1994; Lemerise & Arsenio, 2000). A tendency to over-attribute anger in ambiguous situations is related to higher rates of aggression (e.g., Dodge & Pettit, 2003; Dodge, Pettit, McClaskey & Brown, 1986). There is also evidence that children who are both rejected by peers and aggressive respond to anger cues in the context of ambiguous provocation with greater endorsement of hostile goals and modes of problem-solving (Lemerise, Fredstrom, Kelley, Bowersox, & Waford, 2006; Lemerise, Gregory, & Fredstrom, 2005). Laboratory research using staged "threat" situations also shows that rejected-aggressive children's feelings of anger interfere with socially competent responding (e.g., Dodge & Somberg, 1987; Chapter 14; Hubbard, et al., 2002; Orobio de Castro, Merk, Koops, Veerman, & Bosch, 2005; Orobio de Castro, Slot, Bosch, Koops, & Veerman, 2003).

The tendency to over-attribute anger and to respond to potential anger cues with hostile goals and problem-solving responses in ambiguous social situations may be attributed to a lack of understanding of others' private mental states. To accurately attribute intentionality to others' behaviors and to understand one's own internal feelings, children must be able to predict others' mental states and understand that private mental states can cause emotions (Denham, 1998). To assess children's emotional understanding, many researchers (Bradmetz & Schneider, 2004; Denham, Blair, Schmidt, &

DeMulder, 2002; Hughes & Dunn, 2002) use puppet scenarios in which children view puppets or characters experiencing discrete emotions, mixed emotions (puppet feels two emotions at once), and discrepant feelings (puppet may feel one way but displays a different expression). Understanding the causes of more complex emotions, mixed or discrepant, depends upon the child's developing theory of mind (Wellman, 1990) wherein children realize that people (themselves and others) may show one emotion but really be feeling something else or that people may respond externally in unpredictable ways. Theory of mind ability is present in a rudimentary form in early childhood but develops considerably by school age (Denham, 1998; Hughes & Dunn, 2002). Children's beliefs about emotions, their causes, and their properties constitute one important type of theory of mind knowledge.

In a longitudinal study that assessed developmental changes in 4–7-year-old children's beliefs about the causes of anger (one's own and other's anger), 4-year-olds typically reported that their anger was caused by mothers, whereas older children were more likely to report peers and siblings as provokers of their anger (Hughes & Dunn, 2002). At all ages, children reported interpersonal control as the theme of the anger-eliciting event (i.e., mom made me mad because she wouldn't let me go out to play; my friend made me mad because he was mean to me). In another study, Rieffe, Terwogt, and Cowan (2005) presented a series of stories to 4-, 6-, and 10-year-old children (boy receives gift from his mother; girl goes outside to play hide and seek) and asked them to predict how they thought the character would feel. After making their predictions, which were accurate for the given circumstances, the researchers then told the children that the character actually felt atypically sad, angry, or fearful. Desire references were used by children of all ages to explain atypical sadness and anger; the boy was mad because he did not get the gift he wanted, whereas belief references were used to explain fear.

Thus, preschoolers' growing understanding of emotional situations contributes to their abilities to understand others' intentions and predicts socially competent behavior when they begin school (Denham, 1998; Denham et al., 2002). Children who have problems managing anger are more likely to have deficits in understanding the intentions of others and are more susceptible to hostile attributional biases and associated aggression (especially reactive aggression, see Hubbard, Romano, McAuliffe, & Morrow, this volume), particularly in arousing situations (e.g., Dodge & Somberg, 1987; Orobio de Castro et al., 2005; 2003). Thus, the ability to regulate anger in stressful situations enables children to more accurately understand the intentions of others. However, problems in regulating anger tend to exacerbate difficulties with peers.

13.3 Regulating Anger

Although infants do have some primitive capacity to regulate arousal via orienting to and avoiding stimulation (Mangelsdorf, Shapiro, & Marzolf, 1995), during infancy, caregivers' interventions are crucial in helping the infant regulate arousal, and eventually, in learning to self-regulate arousal. For some infants and caregivers, the regulation of arousal is more challenging due to temperament-based differences in anger-proneness (e.g., Calkins et al., 2002). However, sensitive and responsive caregiving that protects the infant from stress and overstimulation is associated with better long-term outcomes (Blair, 2002; Kochanska et al., 2005). Thus infants' expression and regulation of emotions, including anger, develops in the context of interpersonal interaction. Infants' growing perceptual and cognitive capacities allow them to discriminate familiar caregivers from other individuals and to form expectancies based on past histories of interactions, ultimately culminating in attachment relationships of varying security (Ainsworth, Blehar, Waters, & Wall, 1979). Attachment relationship quality has been shown to affect expression and regulation of emotions. The majority of children form secure attachments with their caregivers; these secure attachment relationships are

associated with increases in the expression of positive emotions and decreases in the expression of negative emotions (including anger) between 14 and 33 months. However, children who form insecure attachments with caregivers continue to express negative emotions; in fact children whose caregivers have been abusive actually increase in the expression of negative emotions, particularly anger (Kochanska, 2001).

For most children, the preschool period is a time for a tremendous growth in the regulation of anger and other emotions. Emotion regulation is multifaceted; it involves learning strategies to modify, modulate, and/or cope with felt emotion as well as learning strategies for displaying emotions appropriate to situations and for constructively coping with emotion-inducing situations. Successful emotion regulation and coping require children to both recognize and understand emotions so that they may employ relevant and appropriate strategies for regulating emotions and coping with challenging situations. The development of language provides a powerful tool for describing and understanding emotional experiences and enhancing communication with caregivers and peers. Improved communication skills contribute to more constructive coping with conflicts over toys and possessions and with frustrations; accordingly, for most children, both temper tantrums and physical aggression decrease over the late preschool and early elementary years (Lemerise & Dodge, 2008; NICHD ECCRN, 2004; Potegal, Kosorok, & Davidson, 2003; Shaw, Gilliom, Ingoldsby, & Nagin, 2003; Tremblay, et al., 1996). Language also is an important socialization tool for parents who can label emotional experiences for children and offer suggestions for coping (i.e., emotion coaching, Gottman et al., 1997 and see below). Between 3 and 5 years, there are important developmental advances in children's understanding of mental states (theory of mind, e.g., Wellman, 1990) and understanding of the causes and situational determinants of emotions in self and others that also contribute to a growing capacity to self-regulate anger and other emotions (Denham, 1998). However, for the minority of children who are less successful in learning how to regulate anger and other strong emotions, negative emotionality and poor emotion regulation predict peer relationship problems and externalizing problems in preschool and elementary school (e.g., Eisenberg et al., 1994, 1997, and see below).

Nancy Eisenberg and colleagues have conducted programmatic research that examines the interaction between children's emotionality and their capacity for regulating emotion and the impact of this interaction on social functioning, both concurrently and longitudinally. Careful observations of preschool-age children have revealed that there are individual differences both in children's emotional intensity and in how they regulate strong emotion, including anger. More constructive (and effective) emotion regulation strategies included shifting attention away from the stressor as well as emotion- and problem-focused coping, whereas less constructive (and less effective) emotion regulation strategies were venting and physical retaliation/aggression (Eisenberg et al., 1994). Children who were high in emotion intensity and who used the less constructive emotion regulation strategies were seen by teachers and parents as less socially skilled. The combination of high emotional intensity and poor emotion regulation in preschool predicted problem behaviors in elementary school. Interestingly high emotional intensity in the context of good regulatory strategies was not associated with these later risks (Eisenberg et al., 1997), which suggests that the risks associated with children's negative emotionality can be offset by parenting that promotes both emotion regulation and learning effective strategies for self-regulation of emotion.

Children's ability to regulate anger depends in part on the early socialization of this skill by their parents. Denham (1993) found that mothers who responded to children's anger with calmness or cheerfulness had children who exhibited more happiness in potentially stressful situations such as the doctor's office, regardless of children's ages, gender, and emotional expressiveness patterns. On the other hand, matching children's anger with parental anger tends to lead to poorer compliance and self-regulation by the child (Kochanska, 1991). As previously discussed, children of all ages tend to

relate unmet desires as underlying thematic causes of anger. Yet as Bugental, Blue, and Lewis (1990) note, parents of children with difficult temperaments, prone to uncooperativeness, tend to withhold desired outcomes from their children in an effort to gain compliance, thus indirectly socializing their children to feel higher levels of negative emotions. Parental socialization strategies that emphasize warmth and open emotional expressiveness contribute to children's overall emotional development including appropriate expression, perception, and regulation of anger (Denham, 1998). However, punitive strategies laced with parental harshness, such as threats and punishment, lead to deficits in all areas of emotional development, placing children at risk in their future relationships with peers (Denham, 1998).

By elementary school, peer group norms emphasize regulation of strong emotions (Lemerise & Dodge, 2008; Parker & Gottman, 1989). Although children, regardless of social status in the peer group, are well aware of peer group norms for controlling anger (Underwood, 1997), they also report that anger is more difficult to control than other emotions (Zeman & Shipman, 1997). Reacting angrily and aggressively toward one's peers puts children at an increased risk of facing peer rejection, peer victimization, or becoming a bully or bully-victim (Eisenberg et al., 2005; Hanish et al., 2004; Salmivalli & Nieminen, 2002). For instance, bully-victims tend to be the most aggressive group and are characterized as being especially deficient in controlling anger (Salmivalli & Nieminen, 2002). For children who are reactively aggressive, interventions which emphasize understanding and controlling anger have been shown to be beneficial (e.g., Lochman, Barry, & Pardini, 2003). Thus, early socialization aimed at aiding children in the management of strong emotions may benefit children's future peer relationships.

Peer interactions, particularly friendship interactions, are an important context for learning to manage strong emotions, including anger. The maintenance of friendships also may be an important source of motivation for regulating anger. Between middle childhood and adolescence, there is evidence from a longitudinal study of friendship that more effective anger regulation strategies come to be used when friends have a conflict. In middle childhood, aggressive and distancing strategies were more common, but these declined in adolescence. Adolescents used negotiation more frequently than they had in middle childhood (von Salisch & Vogelgesang, 2005). These longitudinal data suggest that friendship may be an important context for learning to regulate anger adaptively. Children who are not well accepted by peers and have few or no friends may be deprived of an important socialization context for learning to regulate anger. Other data show that children who have difficulty regulating anger and other emotions are at risk for a variety of problems, including school failure, peer rejection, aggression, and victimization (e.g., Blair, 2002; Denham et al., 2002; Eisenberg, et al., 1997; 2005; Hanish, et al., 2004). In particular, converging evidence shows that children and adolescents who have difficulty regulating anger, who express and/or experience more anger, and/or have an anger perception bias are at high risk for externalizing disorders (e.g., Bohnert, Crnic, & Lim, 2003; Coles, Greene, & Braithwaite, 2002; Keltner, Moffitt, & Stouthamer-Loeber, 2005; Rydell, Berlin, & Bohlin, 2003; Zeman, Shipman, & Suveg, 2002).

13.4 Conclusions

From the preschool years through middle childhood and beyond, children make great strides in the perception and understanding of anger and in regulating the anger they feel and express. These developmental changes are supported by advances in children's perceptual and cognitive development and by sensitive and responsive caregiving. Parenting practices that facilitate children's ability to recognize anger and learn effective strategies to manage anger allow children to manage their arousal and effectively engage with the environment, especially with peers. The developmental tasks

of learning to perceive, understand, and express anger in culturally appropriate ways are complicated by variations in temperamentally based anger-proneness. For some children, frustration and anger are more easily provoked, more intense, more long-lasting, and harder to regulate. Supportive and sensitive caregiving helps all children to learn to cope with frustration and anger, but it is especially crucial for children who are anger-prone. The preschool years are a key time in the socialization of anger as perceptual, cognitive, and language development provide important tools for identifying, understanding, and regulating anger. Converging evidence demonstrates that, by the time of school entry, those children who have not mastered these skills are at risk for peer relation problems, poor adjustment to school, and a variety of externalizing problems.

References

- Ainsworth, M. D. S., Blehar, M., Waters, E., & Wall, S. (1979). *Patterns of attachment*. Hillsdale, NJ: Erlbaum.
- Arsenio, W., Cooperman, S., & Lover, A. (2000). Affective predictors of preschool children's aggression and peer acceptance: Direct and indirect effects. *Developmental Psychology*, 36, 438–448.
- Arsenio, W., & Killen, M. (1996). Conflict-related emotions during peer disputes. *Early Education and Development*, 7, 43–57.
- Barefoot, J. C., Dodge, K. A., Peterson, B. L., Dahlstrom, W. G., & Williams, X. B. (1989). The cook-medley hostility scale: Item content and ability to predict survival. *Psychosomatic Medicine*, 51, 46–57.
- Blair, C. (2002). School readiness: Integrating cognition and emotion in a neurobiological conceptualization of children's functioning at school entry. *American Psychologist*, 57, 111–127.
- Blair, R. J., Colledge, E., Murray, L., & Mitchell, D. G. (2001). A selective impairment in the processing of sad and fearful expressions in children with psychopathic tendencies. *Journal of Abnormal Child Psychology*, 29, 491–498.
- Bohnert, A. M., Crnic, K. A., & Lim, K. G. (2003). Emotional competence and aggressive behavior in school-age children. *Journal of Abnormal Child Psychology*, 31, 79–91.
- Bradmetz, J., & Schneider, R. (2004). The role of the counterfactually satisfied desire lag between false-belief and false-emotion attributions in children age 4–7. *British Journal of Developmental Psychology*, 22, 185–196.
- Bugental, D. B., Blue, J., & Lewis, J. (1990). Caregiver beliefs and dysphoric affect directed to difficult children. *Developmental Psychology*, 26, 631–638.
- Buss, K. A., & Goldsmith, H. H. (1998). Fear and anger regulation in infancy: Effects on the temporal dynamics of affective expression. *Child Development*, 69, 359–374.
- Calkins, S. D., Dedmon, S. E., Gill, K. L., Lomax, L. E., & Johnson, L. M. (2002). Frustration in infancy: Implications for emotion regulation, physiological processes, and temperament. *Infancy*, 3(2), 175–197.
- Carpenter, S., & Halberstadt, A. (2000). Mothers' reports of events causing anger differ across family relationships. *Social Development*, 9, 458–477.
- Casey, R. J., & Schlosser, S. (1994). Emotional responses to peer praise in children with and without a diagnosed externalizing disorder. *Merrill-Palmer Quarterly*, 40, 60–81.
- Caspi, A., Moffitt, T. E., Morgan, J., Rutter, M., Taylor, A., Arseneault, L., et al. (2004). Maternal expressed emotion predicts children's antisocial behavior problems: Using monozygotic twin differences to identify environmental effects on behavioral development. *Developmental Psychology*, 40, 149–161.
- Castelli, F. (2005). Understanding emotions from standardized facial expressions in autism and normal development. *Autism*, 9, 428–449.
- Cole, P. M., Tamang, B. L., & Shrestha, S. (2006). Cultural variations in the socialization of young children's anger and shame. *Child Development*, 77, 1237–1251.
- Cole, P. M., Teti, L. O., & Zahn-Waxler, C. (2003). Mutual emotion regulation and the stability of conduct problems between preschool and early school age. *Development & Psychopathology*, 15, 1–18.
- Cole, P. M., Zahn-Waxler, C., & Smith, K. D. (1994). Expressive control during a disappointment: Variations related to preschoolers' behavior problems. *Developmental Psychology*, 30, 835–846.
- Coles, C. J., Greene, A. F., & Braithwaite, H. O. (2002). The relationship between personality, anger expression, and perceived family control among incarcerated male juveniles. *Adolescence*, 37, 395–409.
- Crick, N. R., & Dodge, K. A. (1994). A review and reformulation of social-information-processing mechanisms of children's social adjustment. *Psychological Bulletin*, 115, 74–101.
- Davies, P. T., & Cummings, E. M. (1995). Children's emotions as organizers of their reactions to interadult anger: A functionalist perspective. *Developmental Psychology*, 31, 677–684.

- Denham, S. A. (1993). Maternal emotional responsiveness and toddlers social-emotional competence. *Journal of Child Psychology & Psychiatry*, 34, 715–728.
- Denham, S. A. (1998). *Emotional development in young children*. New York: Guilford.
- Denham, S. A., Blair, K. A., Schmidt, M., & DeMulder, E. (2002). Compromised emotional competence: Seeds of violence sown early? *American Journal of Orthopsychiatry*, 72, 70–82.
- Denham, S. A., Mitchell-Copeland, J., Strandberg, K., Auerbach, S., & Blair, K. (1997). Parental contributions to preschoolers' emotional competence: Direct and indirect effects. *Motivation and Emotion*, 27, 65–86.
- Dodge, K. A., & Pettit, G. S. (2003). A biopsychosocial model of the development of chronic conduct problems in adolescence. *Developmental Psychology*, 39(2), 349–371.
- Dodge, K. A., Pettit, G. S., McClaskey, C. L., & Brown, M. (1986). Social competence in children. *Monographs of the Society for Research in Child Development* (Serial No. 213, Vol. 51, No. 2).
- Dodge, K. A., & Somberg, D. R. (1987). Hostile attributional biases among aggressive boys are exacerbated under conditions of threat to the self. *Child Development*, 58, 213–224.
- Eisenberg, N., Cumberland, A., & Spinrad, T. L. (1998). Parental socialization of emotion. *Psychological Inquiry*, 9, 241–273.
- Eisenberg, N., Fabes, R. A., Guthrie, I. K., Murphy, B. C., Maszk, P., Holgren, R., et al. (1996). The relations of regulation and emotionality to problem behavior in elementary school children. *Development and Psychopathology*, 8, 141–162.
- Eisenberg, N., Fabes, R. A., Guthrie, I. K., & Reiser, M. (2000). Dispositional emotionality and regulation: Their role in predicting the quality of social functioning. *Journal of Personality & Social Psychology*, 78, 136–157.
- Eisenberg, N., Fabes, R. A., Nyman, M., Bernzweig, J., & Pinuelas, A. (1994). The relations of emotionality and regulation to children's anger-related reactions. *Child Development*, 65, 109–128.
- Eisenberg, N., Fabes, R. A., Shepard, S. A., Murphy, B. C., Guthrie, I. K., Jones, S., et al. (1997). Contemporaneous and longitudinal prediction of children's social functioning from regulation and emotionality. *Child Development*, 68, 642–664.
- Eisenberg, N., Sadovsky, A., Spinrad, T. L., Fabes, R. A., Losoya, S. H., Valiente, C., et al. (2005). The relations of problem behavior status to children's negative emotionality, effortful control, and impulsivity: Concurrent relations and prediction of change. *Developmental Psychology*, 41, 193–211.
- Fabes, R. A., Hanish, L. D., Martin, C. L., & Eisenberg, N. (2002). Young children's negative emotionality and social isolation: A latent growth curve analysis. *Merrill-Palmer Quarterly*, 48, 284–307.
- Fine, S. E., Trentacosta, C. J., Izard, C. E., Mostow, A. J., & Campbell, J. L. (2004). Anger perception, caregivers' use of physical discipline, and aggression in children at risk. *Social Development*, 13, 213–228.
- Goodenough, F. L. (1931). *Anger in young children*. Minneapolis: University of Minnesota Press.
- Gottman, J. M., Katz, L. F., & Hooven, C. (1997). *Meta-emotion: How families communicate emotionally*. Mahwah, NJ: Erlbaum.
- Hanish, L. D., Eisenberg, N., Fabes, R. A., Spinrad, T. L., Ryan, P., & Schmidt, S. (2004). The expression and regulation of negative emotions: Risk factors for young children's peer victimization. *Development & Psychopathology*, 16, 335–353.
- Hayden, E. P., Klein, D. N., & Durbin, C. E. (2005). Parent reports and laboratory assessments of child temperament: A comparison of their associations with risk for depression and externalizing disorders. *Journal of Psychopathology & Behavioral Assessment*, 27, 89–100.
- Herba, C. M., Landau, S., Russell, T., Ecker, C., & Phillips, M. L. (2006). The development of emotion-processing in children: Effects of age, emotion, and intensity. *Journal of Child Psychology & Psychiatry*, 47, 1098–1106.
- Hubbard, I. A., & Coie, J. D. (1994). Emotional correlates of social competence in children's peer relationships. *Merrill-Palmer Quarterly*, 40, 1–20.
- Hubbard, J. A., Romano, L. J., McAuliffe, M. D., & Morrow, M. T. (in press). Anger and the reactive-proactive aggression distinction in childhood and adolescence. In M. Potegal, G. Stemmler, & C. Spielberger (Eds.), *International handbook of anger: Constituent and concomitant biological, psychological, and social processes*. New York: Springer.
- Hubbard, J. A., Smithmyer, C. M., Ramsden, S. R., Parker, E. H., Flanagan, K. D., Dearing, K. F., et al. (2002). Observational, physiological, and self report measures of children's anger: Relations to reactive versus proactive aggression. *Child Development*, 73, 1101–1118.
- Hughes, C., & Dunn, J. (2002). "When I say a naughty word." A longitudinal study of young children's accounts of anger and sadness in themselves and close others. *British Journal of Developmental Psychology*, 20, 515–535.
- Jenkins, J. M. (2000). Marital conflict and children's emotions: The development of an anger organization. *Journal of Marriage & the Family*, 62, 723–736.

- Jenkins, J. M., Shapka, J. S., & Sorenson, A. M. (2006). Teenage mothers' anger over twelve years: Partner conflict, partner transitions and children's anger. *Journal of Child Psychology & Psychiatry*, 47, 775–782.
- Keltner, D., Moffitt, T. E., & Stouthamer-Loeber, M. (2005). Facial expressions of emotion and psychopathology in adolescent boys. In Ekman, P. & Rosenberg, E. (Eds.), *What the face reveals: Basic and applied studies of spontaneous expression using the facial action coding system (FACS)* (pp. 532–550). New York, NY: Oxford University Press.
- Kochanska, G. (1991). Socialization and temperament in the development of guilt and conscience. *Child Development*, 62, 1379–1392.
- Kochanska, G. (2001). Emotional development in children with different attachment histories: The first three years. *Child Development*, 72, 474–490.
- Kochanska, G., Aksan, N., & Carlson, J. J. (2005). Temperament, relationships, and young children's receptive cooperation with their parents. *Developmental Psychology*, 41, 648–660.
- Lemerise, E. A., & Arsenio, W. F. (2000). An integrated model of emotion processes and cognition in social information processing. *Child Development*, 71, 107–118.
- Lemerise, E. A., & Dodge, K. A. (2008). The development of anger and hostile interactions. In M. Lewis, J. M. Haviland, & Barrett, L. F. (Eds.), *Handbook of emotions* (3rd ed., pp. 727–739). New York: Guilford Press.
- Lemerise, E. A., Fredstrom, B. K., Kelley, B. M., Bowersox, A. L., & Waford, R. N. (2006). Do provocateurs' emotion displays influence children's social goals and problem solving? *Journal of Abnormal Child Psychology*, 34, 559–571.
- Lemerise, E. A., Gregory, D. S., & Fredstrom, B. K. (2005). The influence of Provocateurs' emotion displays on the social information processing of children varying in social adjustment and age. *Journal of Experimental Child Psychology*, 90, 344–366.
- Lewis, M., & Ramsay, D. (2005). Infant emotional and cortisol responses to goal blockage. *Child Development*, 76, 518–530.
- Lochman, J. E., Barry, T. D., & Pardini, D. A. (2003). Anger control training for aggressive youth. In Kazdin, A. E. (Ed.), *Evidence-based psychotherapies for children and adolescents* (pp. 263–281). New York, NY: Guilford Press.
- Malatesta, C. Z., Culver, C., Tesman, J. R., & Shepard, B. (1989). The development of emotion during the first two years of life. *Monographs of the Society for Research in Child Development*, 54 (1–2, Serial No. 219).
- Malatesta, C. Z., & Haviland, J. M. (1982). Learning display rules: The socialization of emotion expression in infancy. *Child Development*, 53, 991–1003.
- Mangelsdorf, S. C., Shapiro, J. R., & Marzolf, D. C. (1995). Developmental and temperamental differences in emotional regulation in infancy. *Child Development*, 66, 1817–1828.
- Matsumoto, D., Seung Hee Yoo, SH & Chung, J. The expression of anger across cultures (This book)
- NICHD ECCRN (2004). Trajectories of physical aggression from toddlerhood to middle childhood. *Monographs of the Society for Research in Child Development*, Serial No. 278, Vol 69, No. 4.
- Orbio de Castro, B., Merk, W., Koops, W., Veerman, J. W., & Bosch, J. D. (2005). Emotions in social information processing and their relations with reactive and proactive aggression in referred aggressive boys. *Journal of Clinical Child & Adolescent Psychology*, 34, 105–116.
- Orbio de Castro, B., Slot, N. W., Bosch, J. D., Koops, W., & Veerman, J. W. (2003). Negative affect exacerbates hostile attributions of intent in highly aggressive boys. *Journal of Clinical Child & Adolescent Psychology*, 32, 57–66.
- Parke, R. D. (1994). Progress, paradigms, and unresolved problems: A commentary on recent advances in our understanding of children's emotions. *Merrill-Palmer Quarterly*, 40, 157–169.
- Parker, J. G., & Gottman, J. M. (1989). Social and emotional development in a relational context: Friendship interaction from early childhood to adolescence. In T. J. Berndt and G. W. Ladd (Eds.), *Peer relationships in child development* (pp. 95–131). New York: Wiley.
- Pollak, S. D., & Tolley-Schell, S. A. (2003). Selective attention to facial emotion in physically abused children. *Journal of Abnormal Psychology*, 112(3), 323–338.
- Potegal, M., Kosorok, M. R., & Davidson, R. J. (2003). Temper tantrums in young children: (II) Tantrum duration and temporal organization. *J. Developmental & Behavioral Pediatrics*, 24, 148–154.
- Reuter, M. Genetics of hostility and anger (this volume)
- Rieffe, C., Terwogt, M. M., & Cowan, R. (2005). Children's understanding of mental states as causes of emotions. *Infant and Child Development*, 14, 259–272.
- Root, C. A., & Jenkins, J. M. (2005). Maternal appraisal styles, family risk status and anger biases of children. *Journal of Abnormal Child Psychology*, 33, 193–204.
- Rubin, K. H., Burgess, K. B., Dwyer, K. D., & Hastings, P. (2003). Predicting preschoolers' externalizing behaviors from toddler temperament, conflict, and maternal negativity. *Developmental Psychology*, 39, 164–176.

- Rydell, A., Berlin, L., & Bohlin, G. (2003). Emotionality, emotion regulation, and adaptation in 5- to 8-year old children. *Emotion*, 3, 30–47.
- Saarni, C., Campos, J. J., Camras, L. A., & Witherington, D. (2006). Emotional development: Action, communication, and understanding. In Damon, W. & Lerner, R. M. (Eds.-in-chief), Eisenberg, N. (Vol. Ed.). *Handbook of child psychology, 6th Ed.: volume three: Social, emotional, and personality development* (pp. 226–299). Hoboken, NJ: Wiley.
- Salmivalli, C., & Nieminen, E. (2002). Proactive and reactive aggression among school bullies, victims, and bully-victims. *Aggressive Behavior*, 28, 30–44.
- Schudlich, T. D. D. R., Shamir, H., & Cummings, E. M. (2004). Marital conflict, children's representations of family relationships, and children's dispositions towards peer conflict strategies. *Social Development*, 13, 171–192.
- Schultz, D., Izard, C. E., & Bear, G. (2004). Children's emotion processing: Relations to emotionality and aggression. *Development & Psychopathology*, 16, 371–387.
- Schultz, D., & Shaw, D. S. (2003). Boys' maladaptive social information processing, family emotional climate, and pathways to early conduct problems. *Social Development*, 12, 440–460.
- Shaw, D. S., Gilliom, M., Ingoldsby, E. M., & Nagin, D. S. (2003). Trajectories leading to school-age conduct problems. *Developmental Psychology*, 39, 189–200.
- Shipman, K. L., & Zeman, J. (1999). Emotional understanding: A comparison of physically maltreating and nonmaltreating mother-child dyads. *Journal of Clinical Child Psychology*, 28, 407–417.
- Shipman, K. L., Zeman, J. N., Nesin, A. E., & Fitzgerald, M. (2003). Children's strategies for displaying anger and sadness: What works with whom? *Merrill-Palmer Quarterly*, 49, 100–122.
- Smekens, S., Riksen-Walraven, J. M., & van Bakel, H. J. A. (2007). Multiple determinants of externalizing behavior in 5-year-olds: A longitudinal model. *Journal of Abnormal Child Psychology*, 35, 347–361.
- Snyder, J., Schrepferman, L., McEachern, A., & DeLeeuw, J. *The contribution of child anger and fear, and parental discipline to early antisocial behavior: An integrative model* (This book)
- Snyder, J., Stoolmiller, M., Wilson, M., & Yamamoto, M. (2003). Child anger regulation, parental responses to children's anger displays, and early child antisocial behavior. *Social Development*, 12, 335–360.
- Tremblay, R. E., Boulerice, B., Harden, P. W., McDuff, P., Perusse, D., Pihl, R. O., et al (1996). Do children in Canada become more aggressive as they approach adolescence? In Human Resources Development Canada and Statistics Canada (Eds.), *Growing up in Canada: National longitudinal survey of children and youth* (pp. 127–137). Ottawa: Statistics Canada.
- Underwood, M. K. (1997). Peer social status and children's understanding of the expression and control of positive and negative emotions. *Merrill-Palmer Quarterly*, 43, 610–634.
- Van Ijzendoorn, M. H., & Sagi, A. (1999). Cross-cultural patterns of attachment: Universal and contextual dimensions. In J. Cassidy & P. R. Shaver (Eds.), *Handbook of attachment: Theory, research, and clinical applications* (pp. 713–734). New York: Guilford Press.
- von Salisch, M., & Vogelgesang, J. (2005). Anger regulation among friends: Assessment and development from childhood to adolescence. *Journal of Social and Personal Relationships*, 22, 837–855.
- Vosk, B. N., Forehand, R., & Figueroa, R. (1983). Perception of emotions by accepted and rejected children. *Journal of Behavioral Assessment*, 5, 151–160.
- Wellman, H. M. (1990). *The child's theory of mind*. Cambridge, MA: MIT Press.
- Zeman, J., Penza, S., Shipman, K., & Young, G. (1997). Preschoolers as functionalists: The impact of social context on emotion regulation. *Child Study Journal*, 27, 41–67.
- Zeman, J., & Shipman, K. (1997). Social-contextual influences on expectancies for managing anger and sadness: The transition from middle childhood to adolescence. *Developmental Psychology*, 33, 917–924.
- Zeman, J., & Shipman, K. (1998). Influence of social context on children's affect regulation: A functionalist perspective. *Journal of Nonverbal Behavior*, 22, 141–165.
- Zeman, J., Shipman, K., & Suveg, C. (2002). Anger and sadness regulation: Predictions to internalizing and externalizing symptoms in children. *Journal of Clinical Child and Adolescent Psychology*, 31, 393–398.

Chapter 14

Anger and the Reactive–Proactive Aggression Distinction in Childhood and Adolescence

Julie A. Hubbard, Lydia J. Romano, Meghan D. McAuliffe, and Michael T. Morrow

Abstract Years ago, Averill (1982) stressed that all anger does not result in aggression, and that all aggression is not the result of anger. The second half of this idea, that aggression can have other catalysts besides anger, foreshadows the distinction between reactive and proactive aggression. Reactive aggression is defensive, retaliatory, and in response to real or perceived provocation. Proactive aggression, on the other hand, is displayed to reach a goal, whether that goal involves material or territorial gain or social dominance. This chapter will review and critique existing empirical work demonstrating that anger is positively related to reactive aggression, but not proactive aggression, in children and adolescents. Our review will include both questionnaire-based and laboratory-based studies. We will also include a section on the assessment of reactive and proactive aggression, how this assessment is often confounded with the measurement of anger, and ideas for untangling these constructs.

Johnny and Marcus are on the playground at recess. Johnny is shooting baskets, but Marcus really wants Johnny's basketball so that he can start a game with his friends. Marcus comes up behind Johnny, pushes and trips him, and grabs the ball from his hands when he falls. Johnny gets up, sputtering and red-faced, and lunges at Marcus. Johnny pins Marcus to the ground and hits him hard, completely forgetting about the basketball that rolls away.

This vignette illustrates an important theoretical distinction between two types of aggression, here labeled reactive and proactive. Reactive aggression is defensive, retaliatory, and in response to real or perceived provocation. Proactive aggression, on the other hand, is displayed to reach a goal, whether that goal involves material or territorial gain or social dominance. In the above vignette, Marcus' goal-oriented aggression would be labeled proactive, whereas Johnny's retaliatory aggression would be labeled reactive.

Years ago, Averill (1982) stressed that all anger does not result in aggression, and that all aggression is not the result of anger. The second half of this idea, that aggression can have other catalysts besides anger, foreshadows the distinction between reactive and proactive aggression. This distinction has been recognized for decades by researchers of both human and animal aggression. Many different labels have been used to represent the distinction, including hostile/instrumental, affective/predatory, and effectual/ineffectual. However, in all cases, aggression researchers are referring to the same basic phenomenon, that some instances of aggression are driven by defense and retaliation, whereas other instances are for the purpose of achieving a desired goal. Anger is a critical

J.A. Hubbard (✉)
University of Delaware, Newark, DE 19716, USA
e-mail: jhubbard@psych.udel.edu

component of reactive aggression, but proactive aggression is described as unemotional. In this respect, reactive aggression appears closely linked to the aggression–hostility–anger syndrome discussed by Reuter, by Spielberger, and by Williams in this book.

This chapter will review and critique existing empirical work demonstrating that anger is positively related to reactive aggression, but not proactive aggression, in children and adolescents. Our review will include both questionnaire-based and laboratory-based studies. We will also include a section on the assessment of reactive and proactive aggression, how this assessment is often confounded with the measurement of anger, and ideas for untangling these constructs.

14.1 Questionnaire-Based Studies of Anger and Reactive Versus Proactive Aggression

The hypothesis that reactive but not proactive aggression is related to difficulties with anger and its regulation has garnered support across a number of studies (DeCastro, Merk, Koops, Verrman, & Bosch, 2005; Dodge & Coie, 1987; Little, Brauner, Jones, Nock, & Hawley, 2003; Little, Jones, Henrich, & Hawley, 2003; McAuliffe, Hubbard, Rubin, Morrow, & Dearing, 2007; Price & Dodge, 1989; Raine et al. 2006). These studies demonstrated the distinction in a diversity of samples, including elementary-school children (McAuliffe et al., 2007; Price & Dodge, 1989), lower-class African-American boys (Dodge & Coie, 1987), German adolescents (Little, Brauner et al., 2003; Little, Jones et al., 2003), Dutch behavior-disordered boys (DeCastro et al., 2005), and antisocial adolescents (Raine et al., 2006).

Effects were robust even when anger was assessed using a variety of different methodologies. One study used hypothetical vignettes (DeCastro et al., 2005), and several others used peer nominations on items such as “Who gets angry easily?” (Dodge & Coie, 1987; Little, Brauner et al., 2003; Little, Jones et al., 2003; McAuliffe et al., 2007; Price & Dodge, 1989). Finally, a number of studies used children’s self-report on various rating scales (Little, Brauner et al., 2003; Little, Jones et al., 2003; McAuliffe et al., 2007; Raine et al., 2006). The assessment of the subtypes of aggression in these studies is so essential a topic that we devote an entire section to it near the end of this chapter.

In summary, these studies suggest that reactive but not proactive aggression is linked to difficulties with anger and its regulation. However, in all of the studies reviewed above, anger was assessed as a trait through self- or peer report. None of the studies included observational measures of children’s state of anger or measured the physiological arousal that likely accompanies it. We review studies of these issues next.

14.2 Laboratory-Based Studies of Anger and Reactive Versus Proactive Aggression

Theorists use terms such as “hotheaded” to refer to children engaged in reactive aggression and “cold-blooded” to refer to children engaged in proactive aggression. Thus, episodes of reactive aggression are thought to be characterized by high levels of physiological arousal. In contrast, episodes of proactive aggression are consistent with a profile of low physiological arousal (Dodge, 1991; Dodge & Pettit, 2003; Vitaro & Brendgen, 2005).

To date, only one study of the associations between the subtypes of aggression, state anger, and physiological arousal has been published. This is also the only published investigation of the

relations between reactive and proactive aggression and an *observational* measure of anger. In this project, teacher ratings of reactive and proactive aggression were gathered on 272 second-grade children (Hubbard et al., 2002). In a laboratory procedure, these children then lost a board game to a peer confederate who cheated. Observational data on children's anger expression and physiological data on their skin conductance reactivity (SCR) were collected during each turn of the game (for review of the psychophysiology of anger, see Stemmler, this book). We found that reactive, but not proactive aggression, was positively related to observed anger expression and SCR. Moreover, these relations held not only when SCR and anger expression were aggregated across the game but also in terms of rate of increase over the time span of the game. That is, children higher in teacher-rated reactive aggression had steeper increases in their SCR and anger expression across the game. These increases were not related to proactive aggression.

Furthermore, over the course of the game, higher levels of teacher-rated reactive aggression were associated with stronger turn-by-turn relations between children's observed anger and their SCR, although these relations did not vary by children's level of proactive aggression (Hubbard et al. 2004). Thus, reactive aggression in the classroom was related to a strong moment-by-moment connection between children's anger expression and their physiological arousal in a laboratory-based peer interaction. These findings point to the importance of understanding more about the connection between children's anger expression and their online physiological arousal. Some children may have a harder time keeping their physiological arousal from manifesting itself in observable anger (i.e., they have not mastered the display rules for dissembling their angry feelings), and these difficulties may be related to reactive aggression.

More work is clearly needed in several areas. First, the question of whether proactive aggression is characterized by lack of physiological arousal and anger remains open. Second, teacher ratings of classroom-based reactive aggression were related to physiological arousal and observed anger in a laboratory-based peer-provocation situation, but aggression itself was not elicited or measured in the laboratory context. It is important to know more about whether children's physiological and emotional profiles differ *in-the-moment* when they are engaging in actual episodes of reactive versus proactive aggression.

We have recently pilot-tested procedures designed to meet these goals (Hubbard et al., 2009). Our sample for this pilot work consisted of 36 fourth- and fifth-grade boys and girls from diverse racial/ethnic groups. Three laboratory tasks all involved computer-based picture exchanges with virtual peers designed to provide an opportunity for participants to display either reactive or proactive aggression. In each task, participants prepared computer art pictures while they believed that a virtual peer was preparing his/her own picture in another room. Each participant took part in all three tasks, with a different virtual peer each time. During the reactive aggression tasks, the participant sent his/her picture to the virtual peer, who both criticized it and spoiled it. (Two reactive tasks were used, one involving low provocation from the virtual peer and one involving high provocation.) The participant then had an opportunity to comment on the virtual peer's picture and spoil it if he/she chose to do so. The proactive aggression task involved a similar picture exchange. However, in this case, the virtual peer was not provocative (he/she praised the participant's picture and did not spoil it), but the participant increased his/her chance of winning a chosen prize if he/she spoiled the virtual peer's picture. Thus, the reactive tasks involved peer provocation but no instrumental gain from aggression, whereas the proactive task involved no peer provocation but clear instrumental gain from aggression.

We examined two hypotheses. First, we hypothesized that children's aggression would relate positively to their anger expression during the reactive tasks, but not the proactive task. Support for this hypothesis was found across all three tasks. In both the low- and high-provocation reactive tasks, angry verbal intonations were positively correlated with both behavioral reactive aggression (the

amount the participant spoiled the virtual peer's picture) and verbal reactive aggression. In contrast, as expected, no significant relations between anger and aggression emerged for the proactive task.

Our second hypothesis was that children's aggression would relate positively to their physiological arousal during the reactive tasks, but that these relations would be negative during the proactive task. Strong support emerged for this hypothesis as well. The higher children's SCR and heart rate (HR) were in the reactive tasks, the more likely they were to engage in behavioral and verbal aggression during those tasks. Conversely, the lower children's SCR and HR were in the proactive task, the more likely they were to engage in behavioral and verbal aggression during that task.

These results suggest not only that elevated physiological arousal is a primary mechanism driving reactive aggression, but that proactive aggression is actually marked by a notable absence of physiological arousal. Children with the lowest levels of physiological arousal during the proactive task were the most likely to aggress against the virtual peer in an attempt to improve their chances of winning a desired prize. These data provide the first empirical support of theory suggesting that proactive aggression is literally "cold-blooded," in that it is displayed when children are particularly calm and unaroused. These findings mirror a larger literature suggesting that conduct problems and psychopathy in children and adolescents are associated with low levels of physiological reactivity (see, for reviews and meta-analyses, Lorber, 2004; Ortiz & Raine, 2004; Scarpa & Raine, 1997). Furthermore, our work suggests that this larger literature may be indexing relations between physiological arousal and children's proactive aggressive behavior in particular.

In our view, more laboratory-based assessments of the subtypes of aggression and accompanying physiological and emotional processes are needed. Only through such time- and labor-intensive approaches will we deepen our understanding of the way in which anger and physiology differentially accompany and/or drive episodes of children's reactive and proactive aggression.

14.3 Assessment of Reactive and Proactive Aggression in Children

We turn now to a description and critique of the measurement approaches used throughout these studies, as well as to suggestions for improving our assessment of the subtypes of aggression. In particular, we are concerned by the extent to which existing measures of the subtypes of aggression confound the assessment of reactive aggression with the assessment of anger. Many measures include items intended to index reactive aggression that actually describe anger and not aggressive behavior at all. This confusion between anger and aggression is an issue that has plagued researchers of childhood aggression for decades. In our view, it is critical to remember that all angry feelings do not lead to aggressive actions, that anger is an emotion and aggression is a set of behaviors, and that these two constructs require separate and careful assessment.

14.3.1 The Questionnaire Developed by Dodge and Coie (1987)

Many of the studies reviewed here assessed reactive and proactive aggression using a six-item questionnaire developed by Dodge and Coie in 1987. The rating scale was originally developed for use by teachers, and in five of the ten studies demonstrating relations between anger and reactive aggression, teacher report on this scale was used to assess the subtypes of aggression (DeCastro et al., 2005; Dodge & Coie, 1987; Hubbard et al., 2004; Hubbard et al., 2002; Price & Dodge, 1989). An additional study used the scale as part of an aggregated approach to the assessment of reactive and proactive aggression (McAuliffe et al., 2007); teacher and parent report on the Dodge and Coie

(1987) scale were combined with teacher and parent report on a scale developed by Brown, Atkins, Osborne, and Milnamow (1996), as well as peer nominations.

The Dodge and Coie (1987) scale includes three items indexing reactive aggression: (1) "When this child has been teased or threatened, he/she gets angry easily and strikes back," (2) "This child claims that other children are to blame in a fight and feels like they started the trouble," and (3) "When a peer accidentally hurts this child, such as by bumping into him/her, this child assumes that the peer meant to do it, and then overreacts with anger or fighting." Three other items index proactive aggression: (4) "This child gets other children to gang up on a peer that he/she does not like," (5) "This child uses physical force, or threatens to use force, in order to dominate other children," and (6) "This child threatens or bullies other children in order to get his/her own way."

In many ways, the psychometric properties of the scale are strong; e.g., intrascale correlations and coefficient alphas are high, suggesting strong internal consistency. More impressive were data regarding convergent validity. Observational data on children's reactive and proactive aggression in playgroups over five consecutive days were collected, in addition to teacher ratings of the subtypes of aggression on these six items. Teacher ratings of reactive aggression correlated positively with directly observed reactive aggression, even after teacher ratings of proactive aggression were partialled out; the complementary finding held for proactive aggression. In contrast, the correlations between teacher ratings of each subtype of aggression and observations of the other subtype of aggression were nonsignificant.

However, discriminant validity was weaker. In particular, in the development of the scale, items were retained that had factor loadings of greater than 0.40 on both the reactive and proactive subscales. In addition, in the original study, the eigenvalue of the proactive factor was only 0.74 (by convention, factors with eigenvalues <1.0 can be neglected). Finally, confirmatory factor analyses in three studies have replicated the two-factor structure, with the reactive and proactive items loading onto separate factors. (Fite, Colder, & Pelham, 2006 [parent report]; Poulin & Boivin, 2000 [teacher and parent report]; Smithmyer, Hubbard, & Simons, 2000 [correctional facility staff report]). However, one other study failed to replicate this two-factor structure (Roach & Gross, 2003 [teacher report]).

The most concerning issue, in our view, is that two of the three reactive aggression items explicitly describe anger (#1, #3). This wording is particularly troublesome when this scale is used as a measure of the subtypes of aggression in studies demonstrating relations between reactive aggression and anger, the focus of our review. It is difficult to interpret a finding showing that reactive aggression assessed via the Dodge and Coie (1987) scale is related to anger assessed via another method, when in fact the items in the scale describe anger almost as much as they describe reactive aggression.

This issue is worrisome, because so much of the small literature supporting relations between reactive aggression and anger, including several studies from our own laboratory, is based on the use of this particular questionnaire. The findings that have emerged from these studies, reviewed above, make great theoretical and intuitive sense. However, our faith in these findings would be greatly enhanced if they were replicated with other measures with stronger psychometric properties and wording that avoids reference to anger. We turn now to a description of other such measures.

14.3.2 Other Questionnaire Measures of Reactive and Proactive Aggression

Throughout the years, researchers have attempted to develop other questionnaire measures of the subtypes of aggression, and these measures have been used in a few of the studies reviewed in this chapter. The next measure to emerge was a teacher-rating form by Brown and colleagues (Brown

et al., 1996). The psychometric properties of this scale were somewhat improved over the Dodge and Coie (1987) scale. However, the questionnaire suffered from many of the same difficulties, with several of the reactive aggression items describing anger clearly but never actually mentioning aggressive behavior (e.g., "This child gets mad when he/she doesn't get his/her way," "This child gets mad when corrected," "This child gets mad for no good reason"). In spite of the improved psychometrics, this scale never "caught on" with researchers, who continued to rely on the Dodge and Coie (1987) scale. In fact, only one study reviewed in this chapter utilized this measure in the assessment of the subtypes of aggression (McAuliffe et al., 2007).

In the last 5 years, two new self-report measures of reactive and proactive aggression have appeared. However, because they are so newly developed, these questionnaires have not been used in any studies beyond the initial reports of their development, or, in one instance, an additional paper by the same research group. So, time will tell the psychometric strength of these measures and whether they will become commonly used to assess the subtypes of aggression. For now, we will provide a brief overview of each scale's initial development.

The first measure, developed by Raine and colleagues (Raine et al., 2006), includes 11 items indexing reactive aggression and 12 items indexing proactive aggression. Internal consistency estimates for each subscale were strong, the authors provided compelling evidence that a two-factor model fit the data better than a one-factor model, and they replicated these results across two samples of 16-year-olds. However, concerns with item wording apply to this measure, too. Many of its items do not actually describe aggressive behavior at all, but rather simply anger (e.g., "Reacted angrily when provoked by others," "Gotten angry when frustrated," "Become angry or mad when you don't get your way").

The second adolescent self-report measure, developed by Little and colleagues (Little, Brauner et al., 2003; Little, Jones et al., 2003), is considerably more promising. This measure assesses two forms (overt and relational) and two functions (reactive and proactive) of aggression. Six items assess overt aggression, with no reference to function (e.g., "I'm the kind of person who hits, kicks, or punches others"). Six more items repeat these items, but with the phrase "to get what I want" added, to assess proactive overt aggression. Similarly, six other items repeat the first six items, but this time with phrases such as "When I'm hurt by someone" added to assess reactive overt aggression. The same pattern is followed to create six items each to assess basic relational aggression, proactive relational aggression, and reactive relational aggression. These items were very carefully worded to avoid any mention of anger. To create scores for reactive aggression, reactive overt aggression scores are regressed on to overt aggression scores, and reactive relational aggression scores are regressed on to relational aggression scores; the resulting residuals representing "pure" reactive aggression are then averaged together. The same approach is used to create proactive aggression scores. Little and colleagues collected data on this scale from two large samples of fifth- through tenth-grade German adolescents. In both samples, a model with two forms and two functions of aggression fit the data better than other models, and internal consistency estimates were good. Furthermore, results were replicated across different ages and genders.

Clearly, this scale represents an advance over previous questionnaires, and we are eager to see it used in future studies. However, the fact that the scale has only been developed in a self-report format is a limitation, because it can only be used to assess the subtypes of aggression in older children or adolescents. Little and colleagues claim that only self-report measures are appropriate for determining a behavior's function, because no one but the individual can know why he/she behaved in a particular way. However, we disagree. Observational studies of the subtypes of aggression suggest that independent observers can reliably agree on the function of aggression (Boivin, Dodge, & Coie, 1995; Dodge & Coie, 1987; Hubbard, Dodge, Cillessen, Coie, & Schwartz, 2001; Price & Dodge, 1989), suggesting that parents, teachers, and peers may be able to do so as well. We consider

the development of new teacher-, parent-, and even peer-report measures of reactive and proactive aggression that follow the format developed by Little and colleagues to be an important next step.

14.3.3 Observational or Laboratory-Based Approaches to Measuring Reactive and Proactive Aggression

Finally, two of the studies reviewed here assessed the subtypes of aggression observed in naturally occurring situations or provoked in laboratory paradigms. First, Price, and Dodge (1989) assessed reactive and proactive aggression in kindergarten and first-grade children through observational coding during free play periods at school, as well as with the Dodge and Coie (1987) teacher-rating scale. In this study, peer nominations assessing anger were positively related to both teacher-rated and observational measures of reactive aggression but not to these measures of proactive aggression.

Second, the task recently developed in our laboratory provides children with opportunities to display both reactive and proactive aggression (Hubbard et al., 2008). In this task, participants are given the chance to spoil a virtual peer's picture, sometimes for instrumental gain and sometimes in response to provocation. Behavior-based measures of reactive and proactive aggression (the amount the participant spoils the picture) as well as observationally coded measures of verbal reactive and proactive aggression are collected. In our pilot work, both of these measures of reactive aggression were positively related to observationally coded anger expression and to physiological arousal.

The findings from these observational or laboratory-based studies are important, as are the findings from the investigations by Little and colleagues. Above, we outlined serious concerns about the confounding of anger and reactive aggression in most questionnaire measures of aggression subtypes. Given these concerns, it is difficult to feel sure of the validity of studies demonstrating positive relations between reactive aggression and anger or physiological arousal that have used these questionnaire measures. However, when the subtypes of aggression are assessed using Little and colleagues' measure, in which confounds with anger were carefully avoided, and comparable results emerge, our faith in their validity is greatly enhanced. Studies using observational or laboratory-based approaches to assess the subtypes of aggression provide even further replication and increased credibility.

14.4 In Conclusion

Throughout this review, we have suggested several future directions for researchers of reactive aggression and anger in children to consider. Most important were two recommendations. First, we need more time- and labor-intensive observational and laboratory-based investigations into the subtypes of aggression and anger. Second, we need theoretically derived and psychometrically strong measures of reactive and proactive aggression in children, measures that do not confound reactive aggression with anger. Little and colleagues have paved the way in this regard, but development of teacher-, parent-, and peer-report measures that follow their lead are much needed.

Yet, even with all that we still have to do, we are encouraged by all that we have learned thus far. Credible evidence is growing to suggest that reactive aggression is associated with and/or driven by anger and physiological arousal, but that proactive aggression is marked by a lack of anger and arousal. These findings may seem commonsensical and simply in logical keeping with theory. As our review suggests, though, conducting rigorous research to back up this theory is actually quite challenging. We are making progress, and we will continue to do so, particularly if researchers undertake projects aimed at the goals described above. As evidence grows that reactive aggression

and proactive aggression are driven by very different emotional and physiological processes, our understanding of the nature of childhood aggression will grow as well.

References

- Averill, J. R. . *Anger and aggression: An essay on emotion*. New York: Springer-Verlag.
- Boivin, M., Dodge, K. A., & Coie, J. D. (1995). Individual-group behavioral similarity and peer status in experimental play groups of boys: The social misfit revisited. *Journal of Personality and Social Psychology*, 69(2), 269–279.
- Brown, K., Atkins, M. S., Osborne, M. L., & Milnamow, M. (1996). A revised teacher rating scale for reactive and proactive aggression. *Journal of Abnormal Child Psychology*, 24(4), 473–480.
- De Castro, B. O., Merk, W., Koops, W., Verrman, J. W., & Bosch, J. D. (2005). Emotions in social information processing and their relations with reactive and proactive aggression in referred aggressive boys. *Journal of Clinical Child and Adolescent Psychology*, 34(1), 105–116.
- Dodge, K. A. (1991). The structure and function of reactive and proactive aggression. In D. J. Pepler & K. H. Rubin (Eds.), *The development and treatment of childhood aggression* (pp. 201–218). Hillsdale, NJ: Lawrence Erlbaum.
- Dodge, K. A. & Coie, J. D. (1987). Social information processing factors in reactive and proactive aggression in children's peer groups. *Journal of Personality and Social Psychology*, 53(6), 1146–1158.
- Dodge, K. A. & Pettit, G. S. (2003). A biopsychosocial model of the development of chronic conduct problems in adolescence. *Developmental Psychology*, 39(2), 349–371.
- Fite, P. J., Colder, C. R., & Pelham, W. E. (2006). A factor analytic approach to distinguish pure and co-occurring dimensions of proactive and reactive aggression. *Journal of Clinical Child and Adolescent Psychology*, 35(4), 578–582.
- Hubbard, J. A., Dodge, K. A., Cillessen, A. H. N., Coie, J. D., & Schwartz, D. (2001). The dyadic nature of social information processing in boys' reactive and proactive aggression. *Journal of Personality and Social Psychology*, 80(2), 268–280.
- Hubbard, J. A., Parker, E. H., Ramsden, S. R., Flanagan, K. D., Relyea, N., Dearing, K. F., et al (2004). The relations between observational, physiological, and self-report measures of children's anger. *Social Development*, 13(1), 14–39.
- Hubbard, J. A., Romano, L., McAuliffe, M. D., Morrow, M. T., Rubin, R. M., & Hyde, C. T. (2009). Laboratory-based assessment of children's reactive and proactive aggression and relations to emotions and psychophysiology. Manuscript in preparation.
- Hubbard, J. A., Smithmyer, C. M., Ramsden, S. R., Parker, E. H., Flanagan, K. D., Dearing, K. F., et al (2002). Observational, physiological, and self-report measures of children's anger: Relations to reactive versus proactive aggression. *Child Development*, 73(4), 1101–1118.
- Little, T. D., Brauner, J., Jones, S. M., Nock, M. K., & Hawley, P. H. (2003). Rethinking aggression: A typological examination of the functions of aggression. *Merrill-Palmer Quarterly*, 49(3), 343–369.
- Little, T. D., Jones, S. M., Henrich, C. C., & Hawley, P. H. (2003). Disentangling the “whys” from the “whats” of aggressive behavior. *International Journal of Behavioral Development*, 27(2), 122–133.
- Lorber, M. F. (2004). Psychophysiology of aggression, psychopathy, and conduct problems: A meta-analysis. *Psychological Bulletin*, 130(4), 531–552.
- McAuliffe, M. D., Hubbard, J. A., Rubin, R. M., Morrow, M. T., & Dearing, K. F. (2007). Reactive and proactive aggression: Stability of constructs and relations to correlates. *The Journal of Genetic Psychology*, 167(4), 365–382.
- Ortiz, J. & Raine, A. (2004). Heart rate level and antisocial behavior in children and adolescents: A meta-analysis. *Journal of the American Academy of Child & Adolescent Psychiatry*, 43(2), 154–162.
- Poulin, F. & Boivin, M. (2000). Reactive and proactive aggression: Evidence of a two-factor model. *Psychological Assessment*, 12(2), 115–122.
- Price, J. M. & Dodge, K. A. (1989). Reactive and proactive aggression in childhood: Relations to peer status and social context dimensions. *Journal of Abnormal Child Psychology*, 17(4), 455–471.
- Raine, A., Dodge, K., Loeber, R., Gatzke-Kopp, L., Lynam, D., Reynolds, C., et al (2006). The reactive-proactive aggression questionnaire: Differential correlates of reactive and proactive aggression in adolescent boys. *Aggressive Behavior*, 32(2), 159–171.
- Roach, C. N. & Gross, A. M. (2003). Assessing child aggression: A tale of two measures. *Child & Family Behavior Therapy*, 25(4), 19–38.
- Scarpa, A. & Raine, A. (1997). Psychophysiology of anger and violent behavior. *Psychiatric Clinics of North America*, 20(2), 375–394.

- Smithmyer, C. M., Hubbard, J. A., & Simons, R. F. (2000). Proactive and reactive aggression in delinquent adolescents: Relations to aggression outcome expectancies. *Journal of Clinical Child Psychology*, 29(1), 86–93.
- Vitaro, F. & Brendgen, M. (2005). Proactive and reactive aggression: A developmental perspective. In R. E. Tremblay, W. Hartup, & J. Archer (Eds.), *Developmental origins of aggression* (pp. 178–201). New York, NY: Guilford Press.

Part V
Appraisal and Information Processing in Anger

Chapter 15

Why Do I Get Angry? A Componential Appraisal Approach

Tanja Wranik and Klaus R. Scherer

Abstract Anger is one of the most frequent emotional experiences in normal, everyday life. Surprisingly, however, anger as an emotion still tends to be narrowly defined and poorly understood. In particular, concepts such as anger, hostility, aggression, and frustration are used interchangeably, making scientific research and practical knowledge difficult to integrate. Moreover, even when anger is explicitly defined, often implicit and untested assumptions are made, for example, (1) that anger is directed at another person with the intention to harm him or her, (2) that aggression and hostility are natural consequences (or sometimes precursors) of anger, and (3) that this emotion is associated with poor social integration, health, and well-being. In this chapter, we propose an integrative model for anger. In particular, we will show that a componential appraisal approach to anger is useful for both systematic research and concrete applications. Using this framework, we will argue that anger does not emerge from specific situations or particular environmental or biological factors, but from the way that individuals subjectively give meaning to and evaluate situations or events. Moreover, and contrary to popular conceptions presuming that anger is uncontrollable and/or harmful, we will show that the occurrence and utility of anger can be explained by cognitive processes and individual differences. Finally, we will demonstrate how a componential appraisal approach to emotions allows us to synthesize research on anger as well as the different functional and dysfunctional manifestations of this emotion in everyday life.

Chris is responsible for a research team in a multinational consumer electronics company. He and his team had worked day and night to develop a concept for a new generation of hand held computers and they were confident that the prototype would get favorable management reviews. In addition, his boss, Mark, had promised to support the project. Chris was therefore looking forward to the project presentation meeting, in which management determined which projects would receive additional funding, and which would be asked to stop development. Six projects were presented, and it was clear to the team that the project they proposed was the most advanced and had the best sales projections. When they were later told that Mark had supported two other projects and had agreed to stop developing the hand held computer, most of the team members were surprised. What will Chris feel? How will he react?

T. Wranik (✉)

Swiss National Center for Affective Sciences; Department of Psychology, University of Geneva, Geneva, Switzerland
e-mail: tanja.wranik@unige.ch

15.1 Introduction

Anger is one of the most frequent emotional experiences in normal, everyday life (Scherer, Wranik, Sangsue, Tran, & Scherer, 2004). Correspondingly, a review of the literature suggests that most scientists assume that we know what anger is and why and when it occurs. In fact, however, anger as an emotion tends to be narrowly defined and poorly understood. A closer look at the numerous articles on anger shows that concepts such as anger, hostility, aggression, and frustration are all used interchangeably, making the literature difficult to integrate (e.g., Berkowitz, 1993; Martin & Watson, 1997; Wilkowski & Robinson, 2008; Chapter 11). Moreover, even when anger is explicitly defined, often implicit and untested assumptions are made, for example, (1) that anger is directed at another person with the intention to harm him or her, (2) that aggression and hostility are natural consequences (or sometimes precursors) of anger, and (3) that this emotion is associated with poor social integration, health, and well-being. Fortunately, advances in the affective sciences, as well as the positive psychology movement, are stimulating research and theories which question many a priori assumptions about anger. The contributions to this handbook are also testimony of just how multifaceted the approaches and theories about anger can be, and how new ideas about this emotion stimulate original questions and new methods and challenge preconceived notions.

In this chapter, we propose an integrative model for anger. In particular, we will show that a componential appraisal approach to anger is useful for both systematic research and concrete applications. Using this framework, we will argue that anger does not emerge from specific situations or particular environmental or biological factors, but from the way that individuals subjectively evaluate situations or events. Moreover, and contrary to popular conceptions presuming that anger is uncontrollable and/or harmful, we will show that the occurrence and utility of anger can be explained by cognitive processes and individual differences. Finally, we will demonstrate how a componential appraisal approach to emotions allows us to synthesize research on anger as well as the different functional and dysfunctional manifestations of this emotion in everyday life.

15.2 Different Models of Emotion

As reviewed below, each of the current psychological models of anger explains some aspects of this complex emotion. However, models of anger should help predict why *some* people will experience anger in relation to particular stimuli, situations, or events and why *others* might not react at all or with a very different emotion. Surprisingly, most do not provide a clear answer to this fundamental question.

First, *discrete emotion theories*, based on Tomkins's (1962, 1963) interpretation of Darwin's (1872/1998) account of the evolutionary functions of emotions in man and animals, suggest the existence of *basic emotions*, including anger, sadness, fear, disgust, and happiness, defined as biologically programmed, adaptive responses, characterized by prototypical facial expressions, physiological reactions, and action tendencies, to specific eliciting situations (Ekman, 1992, 1994, 1999, 2004; Izard, 1993; Chapter 21). Anger and fear are characterized as two opposing reactions to threat, which can lead to fight or flight, respectively. Although this approach allows for general predictions as to why anger could occur in response to stereotypical situations (e.g., threat) and what such anger expression may look like (e.g., frowning, scowling, physical aggression), it does not predict individual differences in expression and behavior nor explains why the same situation can lead to a large variety of different emotional feelings and reactions in different individuals on different occasions.

Second, *neo-associationist theory* (Berkowitz & Harmon-Jones, 2004; Chapter 16) is a learning-based model in which aversive stimulation predisposes the individual to negative affect. This in turn

automatically activates feelings, thoughts, memories, and motor impulses, all associatively coupled to an anger or affect-based aggression response. The model provides examples for stimuli or situations likely to activate anger and consequent aggressive behaviors, without specifying detailed elicitation mechanisms or predicting individual differences. For example, the theoretical assumption of spreading activation specifies that thoughts, memories, and feelings are differentially organized within each person's memory, but does not specify how these networks are generated or organized or how one would influence them to change the emotional reaction. The model shares some features with the dimensional model described below and is useful for understanding specific memory components and priming effects underlying anger and aggression; however, it does not allow prediction of anger generation in specific individuals and situations and has little to say about nonaggressive anger behaviors and expressions.

Third, *dimensional models* of emotion focus primarily on the subjective feeling or experience component of emotion. In general, protagonists of this model propose two dimensions, *valence* (feeling good or bad) and *arousal* (degree of felt activation), and suggest that emotions occupy certain regions within this subjective feeling space. For example, Russell (2003) suggests that "core affect" is the primary emotional reaction to a situation or event, characterized by a position in this bi-dimensional space. In this model, anger is characterized by strong, negative valence and high activation values. In addition, the reason a person will report feeling "angry," rather than feeling "bad," "sad," or "guilty," is based on his or her experience, learning, and social norms. Thus, each time an adult labels a child's behavior with an emotion term such as anger or irritation, or children observe these emotion terms being used to label someone else's behavior, they extract information about that instance and integrate this new information with past information associated with the same concept already stored in memory. Throughout their lifetime, individuals will therefore acquire a host of exemplars of what different emotions "feel like" and "look like" and store these as fuzzy categories (Russell & Fehr, 1994). This approach can help explain linguistic origins of emotional categories and the variability in how individuals describe and communicate emotional experiences due to social exposure, observer learning, and formal education. However, it does not specify the underlying mechanisms that determine how individuals acquire these fuzzy categories, is limited to the subjective feelings associated with anger, and has little to say about how these expressive and behavioral components are triggered and organized.

15.2.1 Componential Appraisal Models

Each emotion theory assumes a specific perspective, which may have specific advantages and disadvantages for guiding research and practical application. The main tenet of componential appraisal theories is to explain how and why specific emotions emerge and why not everyone will have the same emotion in response to a given situation.

This approach suggests that the elicitation and differentiation of emotions is based on a process of cognitive evaluations or "appraisals" (e.g., Arnold, 1960; Frijda, 1986, 2005, 2006; Lazarus, 1968; Roseman, 1991; Scherer, 1984, 2001; Ellsworth & Scherer, 2003; Smith & Ellsworth, 1985). The basic notion is that individuals are constantly scanning the environment to make sense of their world and to prepare appropriate actions, if necessary. Moreover, given the variety and complexity of possible stimuli to process, it is important that individuals rapidly determine which ones are relevant for their personal, physical, or psychological well-being. Emotions act as such "relevance detectors" by focusing attention on the appropriate stimuli or event and the ensuing additional subjective evaluations will help predict which emotions are experienced. Thus, individuals will focus their attention only on those situations and events they consider important and relevant for current

goals and well-being, and through a series of appraisal “checks” (described below) will evaluate this relevant situation in a particular way based on past experiences, personality, and current motivations.

In our example, Chris will only experience an emotion if the decision not to fund the project is important to him. If he does not care what happens to the project, or he has other more important things to deal with, he could have no emotional reaction at all. The fact that emotions are only experienced in response to events that are considered *personally important or relevant* explains why other people can have strong emotional reactions to events we consider to be unimportant, or vice versa, and explains why we have a strong emotional reaction in response to something when others think we are over-reacting. In this way, all emotions are *signals* that a particular situation or event is important to the person who experiences the emotion. The *type* of emotion that is experienced will depend on how one evaluates or appraises the event.

15.2.2 Appraisal Processes

Although there is some disagreement on the exact number and type of subjective evaluation “checks” that drive the evaluation process, they can generally be broken down into four general categories (Ellsworth & Scherer, 2003). Below we briefly list these categories, as well as the specific checks within each of them as suggested by the Component Process Model (CPM; Scherer, 1984, 2001). These checks can be schematized as questions individuals ask themselves about a particular stimulus, event, or situation:

1. How relevant is the situation or event for me? (*relevance detection*)
 - Is it novel, sudden, familiar, and predictable?
 - Is it pleasant or unpleasant?
 - Does it directly affect me or my social reference group?
2. What are the implications or consequences of this event and how do they affect my well-being and my immediate or long-term goals? (*implications*)
 - Who is responsible?
 - Did he or she act intentionally?
 - What are the probabilities of different outcomes?
 - Did I expect this to happen?
 - Is this favorable or useful for my current goals or needs?
 - How urgent is it that I take action?
3. How well can I cope with or adjust to these consequences? (*coping potential*)
 - Do I have the necessary control to do something?
 - Do I have the necessary power to translate my strategies into action?
 - Can I deal with the consequences of the situation?
4. What is the significance of this event for my self-concept and to social norms and values? (*normative significance*)
 - How does the situation or event relate to my personal values and principles? (*internal standards*)
 - How will the situation or event influence my status or what other people think of me? (*external standards*)

The CPM (Scherer, 1984, 2001) shares many features with other appraisal theories; however, it uniquely suggests that these appraisal checks will unfold sequentially, in the order specified above. Indeed, based on phylogenetic and ontogenetic reasons, it seems logical that some evaluations will precede others. Thus, as discussed above, detecting which situations and events are relevant is the first step for emotion generation (e.g., we do not experience emotions in response to nonrelevant situations and events); knowing who or what is responsible tends to be useful in determining what the coping options are. For example, if Chris believes that he is responsible for the failed project funding, then he may evaluate his control and power in the situation as high and can, for example, motivate his team to learn from the failure and work harder next time or secure additional training opportunities for him and his group. If he thinks someone else is responsible for the failure, his options will be more complex. Can he blame his boss, Mark, for the failure, and if yes how will this affect his relationship? If he is convinced his project was the best one, how can he learn to accept the unfairness of the decision to stop development? Does he have the necessary skills and opportunities to quit his job or the department and find a better position elsewhere? Several recent empirical studies have confirmed the sequential nature of appraisal dimensions (Aue, Flykt, & Scherer, 2007; Grandjean & Scherer, 2008; Lanctot & Hess, 2007), and ongoing research should provide additional support for the sequential nature of the evaluations in the years to come.

Beyond being sequential, appraisal checks are also dynamic, and the subjective evaluations will usually change several times until a particular emotional event is mastered. Thus, most individuals will experience numerous evaluation cycles within any one, short emotional episode. Given the dynamic, recursive nature of these subjective evaluations, we therefore tend to refer to the overall mechanism as appraisal “processes.”

In our example, Chris probably evaluated the event (not obtaining funds and support for an important project) as *sudden*, *unpleasant*, and *relevant* for his professional well-being. Surprised by not receiving the funds (since his boss had promised to support it and he expected it to be funded), he may have initially evaluated a *lack of control and power* in the situation (the decision was made by other people). Quite rapidly, however, Chris may remember alternative funding opportunities and a conversation with another department head interested in the project. He may evaluate Mark’s failure to support the decision as *unjust* and at the same time realize that he has the *necessary power* and *adjustment* to request funding from another department head. All these evaluations and reevaluations can take place in the space of fractions of a second or minute. If a colleague later asks how Chris felt about not obtaining the funds, he may respond something like: “Oh I don’t know, I felt angry that my boss did not believe in me and this project, and at the same time relieved that I know how he feels about our team. So I guess I am angry at my boss, and at the same time grateful that I finally know that I need to leave his team and work with another group.”

Some critics suggest that appraisals are too cognitive and too slow to account for rapid emotional reactions (see Zajonc’s, 1984, argument for the primacy of an unspecified “affective system”). In retrospect, the use of the term “cognitive” in the 1980s probably created a misleading impression, suggesting that these appraisal checks were verbal, propositional, conscious, and deliberate (Roseman, 1984). Since then, numerous theoretical and empirical papers have addressed this misconception, and new technological advances in the domain of neurology and neuropsychology have also greatly enlarged our definition of cognition (Ellsworth & Scherer, 2003). It should therefore be noted that appraisal processes can occur on several cognitive levels, including sensori-motor, schematic, and propositional processing (Leventhal & Scherer, 1987). Moreover, many emotional situations involve familiar scripts and may elicit bundles of interrelated appraisals. Thus, evaluations can be highly learned and automatic and consequently extremely rapid. For example, Grandjean and Scherer (2008) recently showed that some appraisals can occur within 400 ms after stimulus onset.

Consequently, although sometimes appraisal processes can occur on deliberate and conscious levels, they are more often partially – or totally – nonconscious and automatic. For example, the evaluation process may be slower and more conscious the first time your colleague is late for a meeting, then this lateness becomes a common event.

Given the infinite number of appraisal combinations, there are potentially as many different emotions as there are appraisal outcomes. For the sake of social sharing and communication, however, most individuals categorize their experience into socially and personally constructed categories, and revert to a relatively limited number of specific emotion labels to explain how they subjectively feel. This *subjective feeling* is what most people think of when they talk about emotions. However, subjective feeling is only one of the components of emotion and reflects the person's attempt to explain the rapid and changing evaluations and the ensuing physiological and expressive reactions during the emotional episode. Moreover, since much of the emotion process remains unconscious, subjective feeling will represent only a part of what happened during the emotional episode and will be limited by the individual's linguistic and emotional competence. Since the profile of appraisal results can vary greatly, the feelings will also be extremely diverse (Scherer, 2004), and this helps explain why we can easily spend hours dissecting the situation or event and our evaluations and reactions when talking about emotional episodes. The complexity of the emotional episode is also reflected by the extraordinary variety of anger experiences described by verbal labels, expressions, and metaphors (Averill, 1982; Russell & Fehr, 1994; Lakoff & Kovecses, 1987; Chapter 10).

15.2.3 Why Do I Feel Angry?

As discussed above, emotions are not bounded categories; rather, there are numerous anger-like experiences. Despite the nuances, however, anger experiences often share some general features (Chapter 11). Anger is elicited when an individual evaluates an *important goal* as *obstructed*. Not obtaining funds for a big project will be considered as goal obstructive by most individuals. However, goal obstruction is the basis of many emotions and not unique to anger.¹ Anger is experienced because one believes that an agent *intentionally* obstructed this important goal (e.g., they knew better, they could have done something different, they meant to be hurtful, or the situation could have been avoided). Thus, Chris will be angry at his boss, Mark, if he believes that Mark had *control* of the funds and the *power* to distribute these funds. If funds are distributed using a chance lottery system, or if Chris believes that Mark has very little decisional power, anger at Mark would be less likely. Intentionality attribution also means that anger will usually be directed toward other persons or animals, or toward oneself, because inanimate objects cannot act intentionally. In our example, Chris might be angry at Mark for not believing in the project or for not keeping his promise and might *blame* him for the decision to terminate the project. However, he could also be angry at himself for trusting Mark or for not being more experienced in corporate politics and funding practices. Finally, the internal or external norm of *injustice* and *moral violations* often plays a strong role in anger elicitation. Thus, Chris may feel that Mark treated him and his team unjustly, by lying to them, or by giving the funds to other, apparently less deserving, colleagues.

¹Frustration has been defined as “a barrier to expected goal attainment” (e.g., Berkowitz, 1993). According to the frustration-aggression hypothesis by Dollard, Doob, Miller, Mowrer, and Sears (1936), aggressive behavior can always be traced back to a frustration. In our view, goal obstruction is one of the variables that helps separate “positive” and “negative” emotions. Positive emotions result from goal conducive situations and events, whereas negative emotions usually arise from the obstruction of a goal or need. Thus, although we agree that “frustration” can be often related to anger and aggression, it is not a defining characteristic for anger.

Although the terms used to describe these appraisal categories or checks vary, these predictions share a strong agreement and have been confirmed in numerous studies (Averill, 1982; Lazarus, 1991, 2001; Roseman, 2001; Scherer, 2001; Smith & Ellsworth, 1985; Smith & Lazarus, 1993). Other questions remain. First, it is not clear which appraisal checks are necessary or sufficient for anger (Kuppens, Van Mechelen, Smits & De Boeck, 2003). Second, there is no indication of how different appraisal checks are integrated, and if they all carry the same weight (Scherer, 2004). It could be that some appraisal checks carry more weight for specific emotions and that weighing functions vary between persons and cultures. Finally, it is unclear which appraisal checks best predict the *intensity* of emotion. For example, Mikula, Scherer, and Athenstaedt (1998) showed the central role of injustice for anger intensity and Edwards (1998) found that anger intensity required social and normative appraisals such as feeling socially superior or having low self-esteem.

15.3 What Does Anger Look Like?

15.3.1 *Aggression Is Not Anger*

In our example, many would expect Chris to show his anger through some form of aggressive behavior, such as hitting a door, stomping out of the room, speaking crossly to someone, or even shouting at the boss or at an innocent bystander to vent his anger. Violence and even murder at work are not uncommon anger sequels (Bies & Tripp, 1998; Hershcovis et al., 2007). But how likely is it that Chris will react in the ways described above?

Aggression and violence comprise high individual and social costs; the salient relationship between anger and aggression in both scientific and popular accounts is therefore not surprising. In large part, this relationship stems from biological and evolutionary approaches to anger, which argue that basic emotions are due to phylogenetically evolved, genetically encoded, and universal affect programs and that anger is inherently tied to the fight response in most animals (Tomkins, 1962, 1963; Chapter 16; Chapter 21). Extensive contemporary research, which focuses primarily on facial expressions, has cataloged specific appearance-based descriptions of prototypical anger expressions in static photographs based on the idea that anger signals aggression and aggressive intent. These photographic expressions have shown strong reliability in stereotypical situations, which further strengthens the idea that anger and aggression are strongly linked (Keltner, Ekman, Gonzaga, & Beer, 2003; Levenson, 2003; for more information about this approach, see Chapter 8).

Research conducted with nonhuman animals is limited to observation and interpretation of behavior, and although we do not doubt that *Homo sapiens* are biologically pre-programmed to have and express certain types of emotions in extreme situations, we feel that simply equating anger with aggression and related stereotypical expressions and behaviors is a mistake. Analogies between human emotions and the internal motivational states of other animals are still disputed. Conversely, anger has many complex social and cognitive antecedents and functions, which may make it a uniquely human emotion (Tavris, 1989). Most important, perhaps, aggression is by no means the dominant response to human anger. Adults' narrative accounts included reference to a range of nonaggressive behaviors such as cognitive reappraisal, tension-reduction, and communication (Averill, 1982; Van Coillie, van Mechelen, & Ceulemans, 2006). Similarly, young adults are more likely to engage in nonaggressive than in aggressive behaviors in relation to anger (Van Coillie & Van Mechelen, 2006). Finally, aggression is often an indiscriminate response to any stressful situation or frustration by individuals who have not learned a diversified set of affect regulation skills

and by men who are only allowed a limited set of emotional expressions due to traditional gender roles (Verona & Curtin, 2006). Thus, although anger and aggression are sometimes related, the strength of the relationship may be greatly exaggerated and simply be due to the availability heuristic (Bazerman, 1990). Instances of anger with aggression may provoke stronger and more enduring memory effects than the countless nonaggressive instances of anger. It is therefore just as likely that Chris in our vignette will remain silent, smile (a non-Duchenne smile), or go for a walk than that he will frown or shout.

15.3.2 Are There Typical Facial, Vocal, and Physiological Expressions or Patterns for Anger?

If we disentangle anger from aggression, then how do we know that others are angry? So far, research only provides partial answers, and the large variability in possible expressive behaviors makes research in this domain challenging. Indeed, emotions are not static moments and evaluation processes are dynamic and recursive. Moreover, biological subsystems are highly interdependent and changes in one subsystem will elicit related changes in other subsystems. Thus, a componential patterning approach would predict that concrete evaluation patterns will provoke specific facial, vocal, physiological, and neurological changes related to the evaluation process. In other words, the result of each consecutive evaluation pattern will differentially and cumulatively affect the state of all other subsystems.² Given the complexity of evaluation patterns, it is unlikely that we will be able to detect many prototypical signs of anger expression outside a very controlled laboratory environment.

For example, past research on facial expressions was based on photographs (Ekman & Friesen, 1978). However, static photographs lack ecological validity and cannot account for the dynamic nature of emotional signals. Due to practical and ethical constraints, it is also difficult to empirically examine predictions for anger in real-life settings (Stemmler, 2004). Consequently, facial and vocal parameters are primarily examined using stimulus material in which professional actors portray specific emotions using Stanislaski methods (Banse & Scherer, 1996). Although these studies cannot yield conclusive data for real-life response patterning, the dynamic emotional scenarios which combine body movement with facial and vocal expressions across a large number of actors provide useful information for expressive patterns and processes. Another procedure is to track dynamic facial expressions in computer game simulations or to create dynamic facial stimuli for perception tasks (Wehrle, Kaiser, Schmidt, & Scherer, 2000).

The use of dynamic stimuli to measure anger expressions and anger perception provides new information. First, prototypical anger expressions are rarely found in spontaneous production (for those familiar with the Facial Action Coding System, anger is typically associated with the following specific muscle movements: Action Units 1, 2, 4, 10, and 23, see Ekman & Friesen, 1978). Second, even when the stereotypical facial muscle action units are found, they rarely all occur at the same time and form a static or prototypical expression. Third, the incidences of facial movement typically associated with anger are often lower than expected (for example, AUs 4 and 10; Scherer & Ellgring, 2007). Similarly, other research has shown that facial expressions rarely emerge as prototypical

²The concrete predictions and justification for the relationship between evaluation patterns and specific facial, vocal, physiological, and neurological patterns are described in (Scherer 2001).

clusters, but rather as sequential and fluid processes reflecting the underlying appraisal patterns (for a discussion, see Wehrle et al., 2000). For example, the frown (AU4, corrugator action) is not always found as part of an anger expression, which may consist only of a narrowing of the eyes and a square mouth. Overall, the results confirm that individual facial movements are driven by appraisal patterns. However, they also indicate that although anger expressions contain some emotion-specific information, these are often quite subtle and reflect the variability of appraisal processes.

Interestingly, research and practice show that many people are quite good at “reading” faces and detecting emotions in others, especially of people they know, or those from similar cultural groups (Elfenbein & Ambady, 2002). And yet, empirical evidence concerning what is being read to make the inferences is sparse. The componential appraisal approach suggests that perception of emotions in others probably consists of perceiving ongoing appraisal processes as they emerge through facial movements, eye gazing, and other nonverbal signals. However, more research is needed to determine which indicators are being used to infer appraisals and emotions from others, and why some people are more accurate than others.

Research on vocal expression is relatively rare and we still have much to learn about how anger is produced and perceived in the voice and how anger prosody is related to different evaluation patterns (for an extensive discussion of vocal expression, see Chapter 9). So far we know that hot anger is usually vocally portrayed with a high fundamental frequency (including high-frequency variation), high amplitude, fast tempo, and elevated energy in higher spectrum frequency ranges. A discriminant analysis using these and other vocal parameters for 14 emotions also showed an accuracy score of 0.75 for hot anger (Banse & Scherer, 1996). Strong or hot anger therefore has a unique signature and is easily perceived. Less is known about more subtle forms of anger expressions, which are much more frequent in everyday life. Finally, there are also strong individual differences in generation and perception in both vocal and facial expression, suggesting differences in emotional competence (Mayer & Salovey, 1997).

The systematic search for specific physiological patterns for anger has been going strong since the 1950s (Ax, 1953, Chapter 11) and the hope to localize anger in the brain more recent (Harmon-Jones & Allen, 1998). Despite a number of valiant research efforts, however, the evidence for specific physiological or neurological patterning is rather sparse. In part, this is because of the difficulty to induce anger with sufficient intensity in the laboratory, especially in the highly controlled and artificial settings necessary for physiological and neurological experiments. However, given the individual nature of anger evaluations, the varieties of anger experiences, and the idiosyncratic nature of physiological (and perhaps also neurological) reactions, we assume that understanding these processes will keep the research community busy for many years.

In sum, expressions and physiological reactions related to anger are not stereotypic and static, nor are they exclusively related to aggressiveness. Rather they show some specificity for anger but also indicate large variability. In the next section, we will discuss the adaptive function of this variability.

15.3.3 Anger Expressions Can Be Functional

The search for typical expressive and physiological markers of anger is based on the assumption that individuals will always behave in the same way and will not automatically adjust their emotional expressions to benefit their goals or adjust to the context. And yet both research and everyday experience indicate that people do all kinds of things when they are angry, and that these behaviors do not have to be destructive or harmful.

In agreement with the biological and evolutionary approaches, we believe that emotions generally prepare functional responses. However, we suggest that expressive behaviors are not generally adaptive, but need to be adaptive for a specific individual under particular conditions. Consequently, they must correspond to an individual's assessment of the situation, which explains why anger can lead to a host of related but different expressions and behaviors.³

15.3.4 *Motivational Components or Action Tendencies*

One way to conceive of an adaptive response is to postulate specific action tendencies that are appropriate to the situation as evaluated by the individual. Action tendencies (or *states of action readiness* or *motivational components* of emotion) are major determinants in the relationship between emotional experience and emotional behavior (Arnold, 1960; Frijda, 1986; Frijda, Kuipers, & ter Schure, 1989). Being angry has been associated with numerous action tendencies, including: "I wanted something to not be so, not to exist," "I wanted to go against an obstacle or difficulty, or to conquer it," "I wanted to observe well, to understand, or I paid attention," and "I boiled inside" (Frijda et al., 1989). Other research has shown that anger is related to at least three general behavior categories: (1) *malevolent anger*: "to express dislike," "to break off a relationship," "to gain revenge for the present incident," "to get even for past wrongs" "to bring about a change in the behavior of the instigator for their own good"; (2) *constructive anger*: "to strengthen a relationship," "to assert authority, or improve your image," "to bring about a change in the behavior of the instigator for his or her own good," "to get the instigator to do something for you (which seems to be a mixture of self-centered and altruistic motives)"; and (3) *fractious anger*: letting off steam reactions that are unrelated to the situation. Moreover, these three types of anger behavior categories only accounted for 47% of variance (Averill, 1982). Finally, more recent empirical evidence suggests that constructive anger is much more frequent than malevolent anger, although the latter is closer to what we generally consider to be anger (Weber, 2004).

The idea is that different evaluations of the situation or event lead to different anger experiences which also motivate different types of expressions and behaviors. In our example, imagine that Chris was informed of the funding decision in a group meeting and that he realized that his communication of anger would be harmful to his career. His anger may motivate him to "pay attention" to the reasons for the decision and figure out how to deal with the situation and his boss at another time, when they can discuss face to face. His expression and behaviors might therefore reflect strong concentration (brow frowning) and engagement (attentive gaze). Or else he may evaluate the situation in a way that he is motivated to "go against the obstacle or difficulty" which his boss represents and therefore be confrontational and assertive in his facial and vocal expressions as well as in his actions. Chris will report feeling angry in both situations, but the behaviors we observe will be very different. Whether the evaluation and behaviors are adaptive will depend on the person and the situation. If Mark is open to assertive behavior and accepts confrontation, he may find Chris's behavior commendable

³The purpose of the emotional system is to allow an organism to adapt to the complex environmental challenges it faces. In the history of *Homo sapiens*, some challenges remained consistent, others changed in functional or social, political, and technological developments. It seems reasonable that the emotional system is flexible and will allow *Homo sapiens* to adjust to many complex social and environmental problems. The idea that emotions are functional for prototypical challenges and general set problems would make the system counterproductive in many instances. Thus, our definition of "adaptive" is larger than the strict biological argument that would assume an identical system for all mammals.

and might see if he can reverse the decision. If Mark believes that the boss is an authority figure that should not be questioned, the confrontation could cost Chris his job.

15.3.5 Appraisal Processes Integrate Knowledge About Social Context and Norms

We rely on facial, vocal, and behavioral cues to communicate our emotions and to determine the emotional state of other people. This means that if Chris does not talk about his feelings to anyone, and if he is someone who does not openly show his emotions, his colleagues and boss might not know that he is angry about the decision. Conversely, if he is a very boisterous, expressive person, the people around him might think he is much angrier about the event than he actually is. Despite cautionary words and empirical evidence that expressiveness and sympathetic activation are not always related (Cacioppo et al., 1992), most of us still confidently use the behaviors we observe and infer the intensity and nature of other people's emotional experiences (Chapter 22). Unfortunately, these popular biases about emotions can also be found in the scientific literature.

For example, technological advances and the strong interest in neuropsychology have led to an explosion of work in the domain of anger *perception* and its consequences, primarily adopting a primary or basic emotion perspective (Adolphs, Russell, & Tranel, 1999; Horstmann & Bauland, 2006; Whalen et al., 2001). In particular, because fear and anger facial expressions are thought to be evolutionary-relevant aversive stimuli, they are often used as experimental stimuli to assess responses to threat. The anger expression is taken to mean that the expresser is a direct threat to the perceiver, whereas the fear expression is thought to signal the presence of some threat other than the expresser, perhaps alerting the perceiver to danger in the environment (Adolphs, 1999; Whalen et al., 2001). In both cases, threat is assumed to lead to avoidance behaviors in normal participants. However, when one actually tests the assumption that typical anger and fear expressions lead to avoidance behaviors, one finds that anger expressions may sometimes facilitate avoidance behaviors; however, fear expressions often elicit *approach* behaviors (Marsh, Ambady, & Kleck, 2005). To explain the relationship between fear expressions and approach behaviors, Marsh, Adams, and Kleck (2005) conducted several experiments. They found that individuals expressing fear were perceived as childlike and helpless and incited perceivers to approach and see how they could help. Individuals expressing anger were seen as mature and strong and perceivers hesitate to approach, preferring to leave them alone to deal with the situation themselves. Facial expressions are believed to be the most important channel for emotional communication between humans. It is therefore somewhat surprising that systematic examinations concerning the relationship between facial expressions and appraisals and emotions within the person perceiving the emotion are still relatively rare.

Beyond facial expressions, we have many proscriptive and prescriptive rules about what to do about anger in different situations (Gedes & Callister, 2007; Scott & Steidtmann, 2006) and most people can resort to various reactions and behaviors in relation to anger, depending on the event or context. Within Western cultures, learning to regulate one's emotions is considered an important developmental task, and successful emotion regulation has been associated with satisfying peer relationships (Fabes et al., 1999), social competence (Eisenberg, 2001), and well-being (Larsen & Prizmic, 2004; Hedwig, Hagemann, Seifert, Naumann, & Bartussek, 2004; Smits & Kuppens, 2005; Spielberger, 1972).

There are also stable preferences in the domain of anger regulation. For example, individuals will differentially prefer to generally manage their anger through assertion, rumination, avoidance, diffusion, directing anger toward other people, or by seeking social support. In addition, women are

more likely to believe that distraction will improve their angry feelings, whereas men believe that aggressive behavior will improve their mood (Harris, 1992).

Beyond such preferred anger regulation styles, most people will also adapt their anger expression as a function of the context or event. Research has shown that interpretations of anger expression can differ greatly depending on status, gender, and culture (Tiedens, 2000; Lewis, 2000; Fitness, 2000). The decision to avoid or approach an angered person – or to engage in some other form of action – will also vary based on such considerations (Aquino, Tripp, & Bies, 2006). Thus, an angry boss might bring about fear, but an angry employee might only annoy his superior (see Rosenwein, 1998 for a historical account concerning the role of status in anger expressions and perception since at least the Middle Ages). Consequently, most individuals do not show their anger toward the boss, but will easily express it toward a colleague, spouse, or child (Fitness, 2000). Similarly, to maintain friendships, children will inhibit anger expression more when faced with peers than in front of a parent (Zerman & Garber, 1996). Children also know that their behaviors in real social interactions are very different than in hypothetical situations. Thus, they engage in much more moderate behaviors in peer interactions, such as feeling and expressing less anger, and hiding and dissembling anger for relationship reasons (Parker et al., 2001). Finally, there are interactions between individual differences and context factors. For example, women are less likely to show their anger if they think they will meet the person later, whereas males are less influenced by such context effect (Evers, Fischer, Rodrigues Mosquera, & Manstead, 2005).

In sum, the interpretation and emotional reaction to other people's anger expressions are based on the perceiver's evaluations of the context and characteristics of the person expressing the emotion, which in turn will dictate their behaviors and reactions and any constructive or destructive relationship outcomes of the interaction. Most important, perhaps, the knowledge of how other persons will react to our emotional expressions is an inherent part of our own evaluative processes and the way we will display or communicate our emotions in various contexts (Manstead & Fischer, 2001). Thus, Chris has a good chance of reacting in an appropriate manner in the meeting situation described above (listening attentively or confronting Mark), because he has learned about Mark's beliefs and reactions during their working relationship. Moreover, Chris will probably not even realize that he has integrated this knowledge into his evaluative system, which is driving the expressive response (Mauss, Cook, & Gross, 2006; Mauss, Evers, Wilhelm, & Gross, 2006). The evaluative system integrates this knowledge into the anger experience and drives the various motivational, expressive, and physiological responses throughout the emotional episode.

15.4 Using the Componential Appraisal Approach to Understand Different Anger Experiences: Anger at Others Versus Anger at the Self

Most of the literature on anger suggests that we are usually angry with someone else; however it is also possible to be angry at oneself. How do these two anger experiences differ? Ellsworth and Tong (2006) conducted a systematic examination of *anger at the self* and *anger at another person* using a traditional approach to map profiles between different emotional states (Keltner & Buswell, 1997). They found that feeling angry at the self was a bit like feeling angry at someone else and a bit like feeling ashamed or guilty, but not really like either of these categories. Anger at the self did not include aspects such as fairness or norm violations, which have been found for anger at another person; despite the self-blame component, anger at the self did not include the moral violations typically found for shame and guilt. Most interesting, perhaps, anger at the self was associated with action tendencies very different from either of these supposedly related emotion categories, and

anger at the self was a solitary, rather than social, emotion. Thus, the emotional experience *anger at the self* was not simply a “normal” anger experience with a different angering object but had unique appraisal patterns and action tendencies.

Systematic examinations in our laboratory indicate that anger at the self is usually shorter and less intense than anger at another person and somewhat less frequent in everyday life. Moreover, even if anger at the self is often a solitary emotional experience, the emotion-inducing events or consequences are easily shared with others and tend to motivate the individual to engage in a host of proactive and constructive behaviors, such as improving relationships, studying more, and taking better care of oneself. Attitudes about anger at the self and anger at others in the work context also indicate that anger at the self is seen as significantly more positive, motivating, and functional for work quality and relationships than anger at the other. The causes, processes, action tendencies, and consequences of these two types of anger are significantly different, and yet individuals clearly found that they both had an anger quality (Wranik & Fiori, 2008; Rivera & Wranik, 2008).

Anger is typically considered to be a social emotion because it is usually provoked by or within social interactions (Averill, 1982). Moreover, the more intense the anger experience, the more individuals tend to engage in the act of blaming another person for their emotional experience (Berkowitz, 1990; Quigley & Tedeschi, 1996). This means that if anger is reported or displayed in a social situation, one would assume that the anger is necessarily directed at one or several of the individuals involved. This assumption was tested and found to be only partially correct. Even when clearly engaged in a social interaction, such as an interdependent achievement task, individuals are just as likely to blame themselves and be angry with themselves than they are to blame the interaction partner and be angry with him or her (Wranik & Scherer, 2001, 2008). Going beyond the emotion label anger and examining underlying appraisal dimensions or other components might show that anger experiences can vary greatly in quality and that our assumptions are sometimes wrong.

Anger at the self is relatively frequent and seems to be different from anger at another (Wranik & Fiori, 2008); why have varieties of anger experiences been neglected? When one examines the experimental evidence used to make the theoretical predictions for anger, it becomes evident that other-accountability is often chosen as a predictor of anger because it is *more frequently* associated with anger and not because it is *always* associated with anger. Smith and Ellsworth (1985), for example, originally found that anger plots into the quadrants of *human control* and *other-responsibility control*. These types of results indicate that *compared to other emotions* – anger is more likely to be due to human action and is associated with other-responsibility. In addition, anger at other persons may either be a more frequent occurrence in everyday life than anger at the self or else it may be more readily available in memory. For example, Frijda et al. (1989) gave participants a list of emotions, asked them to remember a time they had experienced that emotion, and then to rate each situation on a series of appraisal dimensions. When thinking about anger, and the events that caused this emotion, it is likely that anger at another person is much more important and has more far-reaching consequences than anger at the self.

Our research suggests that anger at the self is a momentary experience that either activates the person to act, “I know I can do better, and I will do this again in order to receive better results,” or not to act, “I know I should do better, why can’t I get it together?” and the emotion is then quickly transformed to joy and pride or shame and depression depending on the outcome of any action taken or not taken (Wranik, 2005). In addition, to resolve anger, it is often necessary to forgive (Kassinove & Tafrate, 2002; Lawler-Row, Karremans, Scott, Edlis-Matityahou, & Edwards, 2008). Given the actor–observer difference in attribution (Jones & Nisbett, 1972) it is certainly much easier to forgive oneself and move on to new thoughts and emotions than to forgive someone else. Anger at another person is more likely to linger in memory and can grow more intense and damaging through rumination and social sharing (Tavris, 1989; Barber, Maltby, & Macaskill, 2005). These different

processes for anger at the self and anger at other persons could lead to different memory encodings and may explain the frequency of this emotion when individuals are asked to remember thoughts and feelings related to anger, a very common procedure within emotion research. Since we rarely ask about the underlying appraisal processes, we are missing a part of the story, and many studies about “anger” may in fact be studying relatively different emotional experiences. Future research on anger may wish to collect information beyond the standard emotional labels, and in so doing find novel patterns linking emotional experience, expressions, and behaviors for different types of persons, cultures, or situations.

15.5 Individual Differences in Anger Experience

So far we have shown how appraisal processes can lead to a variety of anger experiences and behaviors. We now address the question of why some people experience anger more frequently than others.

Clinical psychologists and anger management counselors confirm that “high trait anger” individuals are more prone to experience and express anger (Kassinove & Tafrate, 2002; Schiraldi & Kerr, 2002; Chapter 18). Reasons for frequent anger (or its unfortunate proxy “aggression”) have suggested numerous factors including genetics (Giegling, Hartmann, Möller, & Rujescu, 2006), personality (Kuppens, 2005), physical illness (Santos, Caeiro, Ferro, Albuquerque, & Figueira, 2006), pain (Chapter 26), mental illness (Chemtob, Novaco, Hamada, Gross, & Smith, 1997; Fraguas et al., 2005; Orth & Wieland, 2006), neurological damage (Ashwin, Wheelwright, & Baron-Cohen, 2006; Graham, Devinsky, & LaBar, 2007; Lawrence, Goerendt, & Brooks, 2007), or developmental and educational factors (Crowell, Evans, & O’Donnell, 1987). The aim of most of this research is to identify groups or types of individuals who might be particularly at risk for developing problems with anger and aggression.

So what is high trait anger? Despite the large and growing amount of literature in the domain of personality and affect (Goldsmith & Davidson, 2004), there is still little information concerning how personality leads to feelings of anger for particular types of individuals in specific situations. This is primarily because personality researchers often use emotional concepts in a very global, undifferentiated fashion or specific to a particular personality theory, thereby making integration of findings from different studies difficult (Averill, 1997). In addition, research on anger has been dominated by the temperamental trait approach and little is known about the underlying processes leading to anger in specific situations (Kuppens, 2005). Finally, much of the research looks at aggressive behaviors and hostility, or at perception of angry faces, rather than at anger feelings. Thus, past research has primarily focused on the frequency of hostility experiences (e.g., anger-prone individuals such as the type A personality; Fontaine, Kulbertus, & Etienne, 1996; or trait anger in the form of neuroticism, Costa & McCrae, 1992), or has examined anger expression and regulation (e.g., Behavioral Anger Response Questionnaire (BARQ), Linden et al., 2003; STAXI; Spielberger, Krasner, & Solomon, 1988).

15.5.1 *Appraisal Biases*

Although there may be some genetic or physiological reasons for individual difference in clinical anger, hostility, or aggressiveness (see Chapters 3 and 27), we will focus on the concept of appraisal biases, which we feel can explain individual differences in “normal,” everyday anger experiences. Indeed, if we accept that emotions are generated and modulated by subjective appraisal processes, it seems logical that individuals will differ in how they selectively focus on specific elements of a

situation or event, how these are cognitively encoded, and how these encodings activate and interact with other cognitions and affects in the overall personality system (Mischel & Shoda, 1995, 1998; Kuppens & van Mechelen, 2007; Vansteelandt & van Mechelen, 2006). Perceptive skills, learning, and cultural values may even render certain evaluations relatively stable, so that some people generally evaluate the world as unjust (Schmitt, 1996); or others systematically look for someone to blame when things go wrong (Seligman, 1986). In other cases, these traits and values may only be activated by specific situational cues, so that blaming someone else only occurs in achievement settings but not in relationship setting. In this framework, trait anger can be understood as chronic accessibility or activation levels of particular cognitions and evaluations under specific conditions. In other words, some individuals have distinct and relatively stable if . . . then . . . patterns that become activated by specific events or under particular circumstances, which can systematically influence particular appraisal dimensions and emotions (see also Stemmler, 1997).

Recent research has shown that high trait and low trait anger individuals have different appraisals in reaction to the same situation (Hazebroek, Howells, & Day, 2001). These may explain why some people generally experience anger more frequently or intensely, or experience certain types of emotions under specific conditions. For example, a perfectionist may chronically overestimate the importance of events; an impatient person may overestimate the urgency of situations; a person sensitive to injustice will evaluate many situations as unjust; and a person with low self-esteem may evaluate many situations as threatening and in need of restorative action (Lazarus, 1991).

The variables in Table 15.1 constitute a selection of individual difference variables that could influence specific appraisal dimensions in the CPM model. Some of the variables are traits from

Table 15.1 Possible relationships between appraisal dimensions and specific individual difference variables

	Appraisal dimension	Possible variables
1.	<i>Relevance detection</i>	
	Novelty	Speed of habituation, extent of inhibition
	Intrinsic pleasantness	Anhedonia
	Goal relevance	Human motivation (e.g., achievement-motivation, affiliation-motivation, McClelland, 1985; Murray, 1938)
2.	<i>Implication assessment</i>	
	Causal attribution	Explanatory style (Peterson et al., 1982; Seligman, 1986)
	Outcome probability check	Optimism–pessimism (LOT-R, Scheier & Carver, 1985)
	Discrepancy from expectation	Openness to experience/conservatism (Costa & McCrae, 1992)
	Goal/need conduciveness	Perfectionism
	Urgency	Realism
3.	<i>Coping potential determination</i>	
	Control	Locus of control (Rotter, 1966); illusion of control
	Power	Self-esteem (Rosenberg, 1965), self-efficacy (Bandura, 1997)
	Adjustment	Openness to experience (Costa & McCrae, 1992); flexibility
4.	<i>Normative significance evaluation</i>	
	(a) Internal standards	Individual human values (Schwartz, 1992)
	(b) External standards	Cultural values (Hofstede, 2001)

the five-factor model of personality (e.g., openness to experience), others are social-cognitive personality traits that measure broader individual differences variables (e.g., self-efficacy, self-esteem, optimism) and still others are individual differences in lower-level cognitive processing (e.g., inhibition, processing speed). The idea is that these individual differences will influence specific appraisal dimensions in a relatively stable manner, and help explain why some people are more likely to experience or report certain types of emotions than others under specific conditions. The following section will explain this model in more detail for anger.

15.5.1.1 Relevance Detection

Emotions usually emerge when individuals evaluate a situation as important and relevant for their well-being. Individual differences in goals and motivation should therefore influence which situations a person will judge as goal relevant and consequently potentially emotional. To test this idea, Smith and Pope (1992) selected individuals who were either high in achievement-motivation or high in affiliation-motivation, and measured their cognitive evaluations and emotions of different events using past memory recall, emotion and appraisal vignettes, and projected imagery tasks. As expected, they found that individuals with high achievement-motivation evaluated achievement-related situations as more motivationally relevant than individuals with low achievement-motivation. Individuals with high affiliation-motivation, however, did not appraise affiliation-related tasks as more goal relevant than individuals with low affiliation-motivation. Assuming that the findings for affiliation-motivation were due to the nature of the induction method, Griner and Smith (2000) studied this variable in a laboratory emotion induction, an interpersonal anger-provoking situation. Participants were selected based on extreme scores in affiliation-motivation and asked to teach a computer program to an unmotivated, incapable, and hostile student, who was in reality an experimental accomplice. As predicted, individuals with high affiliation-motivation appraised the interpersonal aspects of the situation as particularly relevant and important, compared to those with low affiliation-motivation. These two studies provide evidence that the relevance of specific events can in part be explained by stable individual differences. Related research suggests that those who experience a great deal of anger could have a low tolerance for frustration or are particularly sensitive to injustice (Kuppens & Van Mechelen, 2007; Schmitt, 1996). These individuals will see many events as goal obstructive or unjust, which in turn can spark anger in response to many more situations and events than those who have a high tolerance for frustration or are less sensitive to injustice.

15.5.1.2 Implication Assessment

Once the importance or relevance of an event for the self has been established, the individual must evaluate the implications of the event and determine the best course of action. To this end, evaluating who or what caused the event is crucial. External attribution and/or blame have often been related to anger at other persons (e.g., Frijda et al., 1989; Roseman, 2001; Scherer, 2001; Smith & Ellsworth, 1985; Smith & Lazarus, 1993). Individuals who systematically believe that negative events are caused by external agents could therefore experience more anger under specific conditions. To test this idea, individuals were selected on extreme scores on a specific individual difference variable, explanatory style (Peterson et al., 1982; Seligman, 1986), which differentiates individuals who generally attribute negative events to external causes from individuals who generally attribute negative event to internal causes. These two types of individuals then participated in a dyadic performance task, a bogus social intelligence test that ended in failure. It was predicted that individuals who generally attribute negative situations to external causes (Externals) would blame the partner

for poor task performance and therefore report more anger than those who generally attribute negative situations to internal causes (Internals). This hypothesis was partially confirmed. Externals were more likely to blame the partner for the failure than Internals, but they did not report more anger than Internals. A closer look at the evaluations showed that the anger reported by Internals was primarily directed at the self, whereas the anger reported by Externals was often directed at the interaction partner. The frequency of anger was the same for Internals and Externals; however, the anger experienced by these two groups was different (Wranik, 2005; Wranik & Scherer, 2009). Internals were almost exclusively angry at themselves, whereas Externals were primarily angry with the partner. Thus, attribution style systematically influences causal attribution and helps explain the type of anger experienced in response to the same event.

15.5.1.3 Coping Potential

Once individuals have evaluated the situation as relevant and determined the implications and causes, their coping potential evaluations will help differentiate between several different types of emotional experiences and responses. Individual differences in how much power and control they have in a particular situation and how easily they adjust to challenges are among the most studied individual differences in the affective domain, not only primarily in the stress and coping literature but in other contexts as well (e.g., Lazarus & Monat, 1991). In short, most of the past research has found that there are individuals who are generally more likely to believe in their capacity to engage in proactive action and deal with different kinds of challenges than others. These individuals believe in themselves and/or in their general capacity to find a solution in most situations (e.g., self-esteem, Rosenberg, 1965; self-efficacy, Bandura, 1997). With this proactive and problem-oriented approach to dealing with situations, these individuals tend to be optimistic about the future and to have more positive emotions (Seligman, 1998). They are also more likely to experience anger, given their general belief that human action can influence events and that they have a right to their emotions (Scherer et al., 2006), although this can depend on cultural values (Mondillon et al., 2005). Inversely, individuals who feel like they cannot control the situations and events around them and have to “succumb” to the world are often faced with helplessness, hopelessness, and depression (Judge & Bono, 2001).

15.5.1.4 Normative Significance

Finally, most individuals also evaluate the significance of events in terms of their self-concept, social norms, and values. Some individuals are more concerned with meeting internal standards, whereas for others it is more important that actions meet with the approval from others, or external standards. Partially, these differences are due to cultural norms, but other variables such as being conservative can also play a role. For example, collectivist cultures focus more on the integration of the individual into the group. Social pressure will therefore be very efficient in obtaining conformity, and individuals are more likely to experience shame if they fail to meet the expectations of others. In more individualist countries, individuals are more likely to experience guilt for not living up to internal standards (Hofstede, 2001; Mesquita & Walker, 2003). Strength and types of perceived norm violations and their relationship to anger depend on factors such as culture, relationship, and gender (Ohbuchi et al., 2004). Other individual differences variables may also be important in explaining consistent appraisal biases in normative significance. For example, perfectionists more often perceive their internal standards as unmet (Hawley, Zuroff, & Blatt, 2006) and could be frequently angry at themselves, whereas sociopaths may have no scruples whatsoever about any of their actions (Klass, 1980) and be angry at anyone who dares question their behavior.

In this section, we illustrated how the concept of appraisal biases can help explain individual differences in anger experience. The idea is that high trait anger individuals have an overactive or faulty evaluation system. They see injustice everywhere, easily find fault with others, etc. Using this approach, both researchers and clinicians can systematically examine evaluation processes and identify the appraisal biases underlying a person's specific reasons for frequent anger. Once the reasons for anger are clear, it should become easier to manage and use anger effectively. For example, imagine that Chris has insight into his sensitivity to injustice and that he often jumps to conclusions concerning other person's intents and motivation. In this case, he could quickly doubt his initial evaluation of the situation and wonder if something else happened in the meeting which caused Mark to vote against their project. Rather than be consumed by the anger, he would try to obtain more information and give Mark the benefit of the doubt until they had time to speak about the situation.

15.6 Conclusions

In this chapter, we proposed an integrative model for anger. We do not claim to explain all aspects of anger, but we tried to provide a framework for research and reflection that we consider both comprehensive and flexible. Indeed, a componential appraisal approach to anger allows researchers to test different hypotheses and ideas about anger, systematically examine individual differences in anger experiences and behaviors, and to think about when and how anger can be functional or dysfunctional. Not all components or processes can be measured in any one experiment. However, we urge researchers to discuss their methods and results only in terms of the processes and component they actually measured and not draw conclusions about the remaining components or underlying process. If research on anger could be grouped into domains – such as anger processes, perception of anger, anger expression, regulation of anger, aggressive behaviors related to anger – then we would know which part of overall emotional phenomenon we are looking at and could more easily integrate new findings into an overall understanding of anger.

15.6.1 *Practical Implications*

Probably the most important contribution of this approach is that anger does not emerge from specific situations or particular environmental or biological factors, but from the way that individuals subjectively evaluate situations or events. This means that each person is responsible for his or her own anger experiences.⁴ Moreover, since we only have emotions in response to events and situations we consider personally relevant and important, experiencing any emotion – and including anger – is first and foremost an indication that we are faced with a situation or an event that is important to us (our health and well-being or the health and well-being of people we care about). Thus, emotions are important signals that can and should be used to motivate our behavior to deal appropriately with the challenge or opportunity at hand. The reason we experience anger, rather than another emotion, is due to the specific way in which we evaluate the situation or event. Common evaluations that lead to anger include the belief that someone or something intentionally obstructed an important goal

⁴This could strongly influence crime of passion verdicts, in which defendants are given mitigating circumstances due to their emotion (see Chapter 22 by Potegal, this volume).

(e.g., they knew better, they could have done something different, they mean to be hurtful or treat us unjustly, or the situation could have been avoided) and that we want things to change.

Moreover, and contrary to the popular conception that anger is destructive or harmful, anger has many useful functions and individuals can learn to harness the emotion for their own needs and goals. For example, we can question whether the event or situation is really that important to us (and if not, we can let go and move onto something else). Or else we can challenge our beliefs and evaluations of the situation that is causing us to feel anger. For example, maybe the person we are angry with did not know that his or her action was harmful for our needs or goal, maybe there was a misunderstanding. By questioning our evaluations or checking our beliefs with the persons implicated, we can perhaps rapidly resolve the situation and learn something new. And finally, even if we are convinced that our evaluations of the situation are correct, and we believe we have a right to feel angry, there is a big difference between being angry and doing something with this anger (see also, Cantor, 1990). Research suggests that there are large individual differences in anger experiences and motivations and that people engage in a variety of expressions and behaviors in response to anger in different contexts. In addition, these expressions and behaviors are often constructive and adaptive. Thus, the idea that anger must necessarily lead to specific reactions, such as aggression, is not supported.

In conclusion, experiencing anger is normal and tends to signal our need or desire for change. However, the way we express our anger, or how we behave in any given situation, can be developed and learned so that our need for change can be as constructive and realistic as possible. Effective emotion regulation is the key to satisfying relationships and long-term health and well-being, and the more we learn about emotions, the more constructive and adaptive our regulation can become (Wranik, Feldman Barrett, & Salovey, 2006). Given that anger is one of the most frequent emotions in everyday life, it might make sense to learn as much as possible about this complex and important emotion.

References

- Adolphs, R., Russell, J. A., & Tranel, D. (1999). A role for the human amygdala in recognizing emotional arousal from unpleasant stimuli. *Psychological Science*, *10*, 167–171.
- Aquino, K., Tripp, T. M., & Bies, R. J. (2006). Getting even or moving on? Power, procedural justice, and types of offenses as predictors of revenge, forgiveness, reconciliation, and avoidance in organizations. *Journal of Applied Psychology*, *91*, 653–668.
- Arnold, M. B. (1960). *Emotion and personality* (Vols. 1 & 2). New York: Columbia University Press.
- Ashwin, C., Wheelwright, S., & Baron-Cohen, S. (2006). Finding a face in the crowd: Testing the anger superiority effect in Asperger syndrome. *Brain and Cognition*, *61*, 78–95.
- Aue, T., Flykt, A., & Scherer, K. R. (2007). First evidence for differential and sequential efferent effects of goal relevance and goal conduciveness appraisal. *Biological Psychology*, *74*, 347–357.
- Averill, J. R. (1982). *Anger and aggression: An essay on emotion*. New York: Springer.
- Averill, J. R. (1997). The emotions: An integrative approach. In R. Hogan, J. Johnson, & S. Briggs (Eds.), *Handbook of personality psychology* (pp. 513–542). San Diego, CA: Academic Press.
- Ax, A. F. (1953). The physiological differentiation between fear and anger in humans. *Psychosomatic Medicine*, *15*, 433–442.
- Bandura, A. (1997). *Self-efficacy: The exercise of control*. New York: Freeman.
- Banase, R., & Scherer, K. R. (1996). Acoustic profiles in vocal expression. *Journal of Personality and Social Psychology*, *70*, 614–636.
- Barber, L., Maltby, J., & Macaskill, A. (2005). Angry memories and thoughts of revenge: The relationship between forgiveness and anger rumination. *Personality and Individual Differences*, *39*, 253–262.
- Bazerman, M. H. (1990). Making rational and irrational decisions. In M. H. Bazerman (Ed.), *Managerial decision making* (pp. 199–227). New York: Wiley.
- Berkowitz, L. (1990). On the formation and regulation of anger and aggression: A cognitive-neoassociationistic analysis. *American Psychologist*, *45*, 494–503.

- Berkowitz, L. (1993). *Aggression: Its causes, consequences, and control*. Boston: McGraw.
- Berkowitz, L., & Harmon-Jones, E. (2004). Toward an understanding of the determinants of anger. *Emotion*, 4, 107–130.
- Bies, R., & Tripp, T. (1998). Revenge in organizations: The good, the bad, and the ugly. In R. Griffin, A. O'Leary-Kelly, & J. Collins (Eds.), *Dysfunctional behavior in organizations: Non-violent dysfunctional behavior* (pp. 49–67). Stanford, CT: JAI Press.
- Cacioppo, J. T., Uchino, B. N., Crites, S. L., Snyersmith, M. A., Smith, G., & Berntson, G. G. (1992). Relationship between facial expressiveness and sympathetic activation in emotion: A critical review, with emphasis on modeling underlying mechanisms and individual differences. *Journal of Personality and Social Psychology*, 62, 110–128.
- Cantor, N. (1990). From thought to behavior: "Having" and "doing" in the study of personality and cognition. *American Psychologist*, 45, 735–750.
- Chemtob, C. M., Novaco, R. W., Hamada, R. S., Gross, D. M., & Smith, G. (1997). Anger regulation deficits in combat-related posttraumatic stress disorder. *Journal of Traumatic Stress*, 10, 17–36.
- Costa, P. T., & McCrae, R. R. (1992). *Manual for the Revised NEO Personality Inventory (NEO-PI-R)*. Odessa, FL: Psychological Assessment Resources.
- Crowell, D. H., Evans, I. M., & O'Donnell, C. R. (1987). *Childhood aggression and violence*. New York: Plenum.
- Darwin, C. (1872/1998). *The expression of the emotions in man and animals* (3rd ed.). New York: University of Chicago Press.
- Dollard, J., Doob, L. W., Miller, N. E., Mowrer, O. H., & Sears, R. R. (1936). *Frustration and aggression*. New Haven, CT: Yale University Press.
- Edwards, P. (1998). *Etude empirique de déterminants de la différenciation des émotions et de leur intensité*. [An empirical study of the determinants of the differentiation and the intensity of the emotions.] Ph.D. Thesis. University of Geneva, Switzerland.
- Eisenberg, N. (2001). The core and correlates of affective social competence. *Social Development*, 10, 120–124.
- Ekman, P. (1992). Are there basic emotions? *Psychological Review*, 99, 550–553.
- Ekman, P. (1994). All emotions are basic. In P. Ekman & R. J. Davidson (Eds.), *The nature of emotion: Fundamental questions* (pp. 15–19). New York: Oxford University Press.
- Ekman, P. (1999). Facial expressions. In M. J. Power & T. Dalgleish (Eds.), *Handbook of cognition and emotion* (pp. 301–320). New York: John Wiley & Sons.
- Ekman, P. (2004). What we become emotional about. In A. Fischer, A. S. R. Manstead, & N. Frijda (Eds.), *Feelings and emotions: The Amsterdam symposium* (pp. 119–135). New York: Cambridge University.
- Ekman, P., & Friesen, W. V. (1978). *Facial action coding system: A technique for the measurement of facial movement*. Palo Alto, CA: Consulting Psychologist Press.
- Elfenbein, H. A., & Ambady, N. (2002). Is there an in-group advantage in emotion regulation? *Psychological Bulletin*, 128, 243–249.
- Ellsworth, P. C., & Scherer, K. R. (2003). Appraisal processes in emotion. In R. J. Davidson, K. R. Scherer, & H. Goldsmith (Eds.), *Handbook of the affective sciences* (pp. 572–595). New York and Oxford: Oxford University Press.
- Ellsworth, P. C., & Tong, E. M. W. (2006). What does it mean to be angry at yourself? Categories, appraisals, and the problem of language. *Emotion*, 6, 572–586.
- Evers, C., Fischer, A. H., Rodriguez Mosquera, P. M., & Manstead, A. S. R. (2005). Anger and social appraisal: A "spicy" sex difference? *Emotion*, 5, 258–266.
- Fabes, R., Eisenberg, N., Jones, S., Smith, M., Guthrie, I., & Poulin, R. (1999). Regulation, emotionality, and preschoolers' socially competent peer interactions. *Child Development*, 70, 432–442.
- Fitness, J. (2000). Anger in the workplace: An emotion script approach to anger episodes between workers and their superiors, co-workers, and subordinates. *Journal of Organizational Behavior*, 21, 147–162.
- Fontaine, O., Kulbertus, H., & Etienne, A. M. (1996). *Stress et cardiologie*. Paris: Masson.
- Fraguas, R., Papakostas, G. I., Mischoulon, D., Bottiglieri, T., Alpert, J., & Fava, M. (2005). Anger attacks in major depressive disorder and serum levels of homocysteine. *Journal of Biological Psychiatry*, 60, 270–274.
- Frijda, N. H. (1986). *The emotions*. New York: Cambridge University Press.
- Frijda, N. H. (2005). Emotion experience. *Cognition and Emotion*, 19, 473–497.
- Frijda, N. H. (2006). *The laws of emotion*. Mahwah, NJ: Lawrence Erlbaum Associates.
- Frijda, N. H., Kuipers, P., & ter Schure, E. (1989). Relations among emotion, appraisal, and emotional action readiness. *Journal of Personality and Social Psychology*, 57, 212–228.
- Geddes, D., & Callister, R. R. (2007). Crossing the line(s): A dual threshold model of anger in organizations. *Academy of Management Review*, 32, 721–746.
- Giegling, I., Hartmann, A. M., Möller, H. J., & Rujescu, D. (2006). Anger- and aggression-related traits are associated with polymorphisms in the 5-HT-2A gene. *Journal of Affective Disorders*, 96, 75–81.

- Goldsmith, H. H., & Davidson, R. J. (2004). Disambiguating the components of emotion regulation. *Child Development*, 75, 361–365.
- Graham, R., Devinsky, O., & LaBar, K. S. (2007). Quantifying deficits in the perception of fear and anger in morphed facial expression after bilateral amygdala damage. *Neuropsychologia*, 45, 42–54.
- Griner, L. A., & Smith, C. A. (2000). Contributions of motivational orientation to appraisal and emotion. *Personality and Social Psychology Bulletin*, 26, 727–740.
- Grandjean, D., & Scherer, K. R. (2008). Unpacking the cognitive architecture of emotion processes. *Emotion*, 8, 341–351.
- Harmon-Jones, E., & Allen, J. J. B. (1998). Anger and frontal brain activity: EEG asymmetry consistent with approach motivation despite negative affective valence. *Journal of Personality and Social Psychology*, 74, 1310–1316.
- Harris, M. (1992). Beliefs about how to reduce anger. *Psychological Reports*, 70, 203–210.
- Hawley, L. L., Ho, M. H. R., Zuroff, D. C., & Blatt, S. J. (2006). The relationship of perfectionism, depression, and therapeutic alliance during treatment for depression: Latent difference score analysis. *Journal of Consulting and Clinical Psychology*, 74, 930–942.
- Hazebroek, J. F., Howells, K., & Day, A. (2001). Cognitive appraisals associated with high trait anger. *Personality and Individual Differences*, 30, 31–45.
- Hedwig, J., Hagemann, D., Seifert, J., Naumann, E., & Bartussek, D. (2004). On the selective relation of frontal cortical asymmetry and anger-out versus anger-control. *Journal of Personality and Social Psychology*, 87, 926–939.
- Hershcovis, M. S., Turner, N., Barling, J., Arnold, K. A., Dupré, K. E., Inness, M., et al (2007). Predicting workplace aggression: A meta-analysis. *Journal of Applied Psychology*, 92, 228–238.
- Hofstede, G. (2001). *Culture's consequences*. Thousand Oaks: Sage.
- Horstmann, G., & Bauland, A. (2006). Search asymmetries with real faces: Testing the anger superiority effect. *Emotion*, 6, 193–207.
- Izard, C. E. (1993). Four systems for emotion activation: Cognitive and noncognitive processes. *Psychological Review*, 100, 68–90.
- Jones, E. E., & Nisbett, R. E. (1972). *The actor and the observer: Divergent perception on causes for behavior*. Morristown, NJ: General Learning Press.
- Judge, T. A., & Bono, J. E. (2001). Relationship of core self-evaluations traits – self-esteem, generalized self-efficacy, locus of control, and emotional stability – with job satisfaction and job performance: A meta-analysis. *Journal of Applied Psychology*, 86, 80–92.
- Kassinove, H., & Tafrate, R. C. (2002). *Anger management: The complete treatment guide for practitioners*. Atascadero, CA: Impact.
- Keltner, D., & Buswell, B. N. (1997). Embarrassment: Its distinct form and appeasement functions. *Psychological Bulletin*, 122, 250–270.
- Keltner, D., Ekman, P., Gonzaga, G. C., & Beer, J. (2003). Facial expression of emotion. In H. H. Goldsmith, R. J. Davidson, & K. R. Scherer (Eds.), *Handbook of affective sciences* (pp. 411–559). New York: Oxford University Press.
- Klass, E. T. (1980). Cognitive appraisal of transgression among sociopaths and normals. *Cognitive Therapy and Research*, 4, 353–367.
- Kuppens, P. (2005). Interpersonal determinants of trait anger: Low agreeableness, perceived low social esteem, and the amplifying role of the importance attached to social relationships. *Personality and Individual Differences*, 38, 13–23.
- Kuppens, P., & van Mechelen, I. (2007). Interactional appraisal models for the anger appraisals of threatened self-esteem, other blame, and frustration. *Cognition and Emotion*, 21, 56–77.
- Kuppens, P., Van Mechelen, I., & Rijmen, F. (in press). Towards disentangling sources of individual differences in appraisal and anger. *Journal of Personality*.
- Kuppens, P., Van Mechelen, I., Smits, D. J. M., & De Boeck, P. (2003). The appraisal basis of anger: Specificity, necessity and sufficiency of components. *Emotion*, 3, 254–269.
- Lakoff, G., & Kovecses, Z. (1987). The cognitive model of anger inherent in American English. In N. Quinn & D. Holland (Eds.), *Cultural models in language and thought* (pp. 195–221). New York: Cambridge University Press.
- Lancot, N., & Hess, U. (2007). The timing of appraisals. *Emotion*, 7, 207–212.
- Larsen, R. J., & Prizmic, Z. (2004). Affect regulation. In R. F. Baumeister & K. D. Vohs (Eds.), *The handbook of self-regulation: Research, theory, and applications* (pp. 40–61). New York: Guilford Press.
- Lawrence, A. D., Goerendt, I. K., & Brooks, D. J. (2007). Impaired recognition of facial expression of anger in Parkinson's disease patients acutely withdrawn from dopamine replacement therapy. *Neuropsychologia*, 45, 65–74.

- Lawler-Row, K. A., Karremans, J. C., Scott, C., Edlis-Matityahou, M., & Edwards, L. (2008). Forgiveness, physiological reactivity and health: The role of anger. *International Journal of Psychophysiology*, 68, 51–58.
- Lazarus, R. S. (1968). Emotions and adaptation: Conceptual and empirical relations. In W. J. Arnold (Ed.), *Nebraska symposium on motivation* (pp. 175–266). Lincoln: University of Nebraska Press.
- Lazarus, R. S. (1991). *Emotion and adaptation*. New York: Oxford University Press.
- Lazarus, R. S. (2001). Relational meaning and discrete emotions. In K. R. Scherer, A. Schorr, & T. Johnstone (Eds.), *Appraisal processes in emotion: Theory, methods, research* (pp. 37–67). New York: Oxford University Press.
- Lazarus, R. S., & Monat, A. (Eds.). (1991). *Stress and coping: An anthology* (3rd ed.). New York: Columbia University Press.
- Levenson, R. W. (2003). Blood, sweat, and fears: The autonomic architecture of emotion. In F. B. M. de Waal, P. Ekman, J. J. Campos, & R. J. Davidson (Eds.), *Emotions inside out: 130 years after Darwin's: The expression of the emotions in man and animals* (pp. 348–366). New York: New York University Press.
- Leventhal, H., & Scherer, R. K. (1987). The relationship of emotion to cognition: A functional approach to a semantic controversy. *Cognition and Emotion*, 1, 3–28.
- Lewis, K. M. (2000). When leaders display emotion: How followers respond to negative emotional expression of male and female leaders. *Journal of Organizational Behavior*, 21, 221–234.
- Linden, W., Hogan, B. E., Rutledge, T., Chawla, A., Lenz, J. W., & Leung, D. (2003). There is more to anger coping than “on” or “out”. *Emotion*, 3, 12–29.
- Manstead, A. S. R., & Fischer, A. H. (2001). Social appraisal: The social world as object of and influence on appraisal processes. In K. R. Scherer, A. Schorr, & T. Johnstone (Eds.), *Appraisal processes in emotion: Theory, methods, research* (pp. 221–232). New York and Oxford: Oxford University Press.
- Marsh, A. A., Adams, R. B., & Kleck, R. (2005). Why do fear and anger look the way they do? Form and social function in facial expressions. *Personality and Social Psychology Bulletin*, 31, 73–86.
- Marsch, A. A., Ambady, N., & Kleck, R. E. (2005). The effects of fear and anger facial expressions on approach- and avoidance-related behaviors. *Emotion*, 5, 119–124.
- Martin, R., & Watson, D. (1997). Style of anger expression and its relation to daily experience. *Personality and Social Psychology Bulletin*, 23, 285–294.
- Mauss, I. B., Cook, C. L., & Gross, J. J. (2006). Automatic emotion regulation during anger provocation. *Journal of Experimental Social Psychology*, 43, 698–711.
- Mauss, I. B., Evers, C., Wilhelm, F. H., & Gross, J. J. (2006). How to bite your tongue without blowing your top: Implicit evaluation of emotion regulation predicts affective responding to anger provocation. *Personality and Social Psychology Bulletin*, 32, 589–602.
- Mayer, J. D., & Salovey, P. (1997). What is emotional intelligence? In P. Salovey & D. Sluyter (Eds.), *Emotional development and emotional intelligence: Educational implications* (pp. 3–31). New York: Basic Books.
- McClelland, D. (1985). *Human motivation*. New York: Cambridge University Press.
- Mesquita, B., & Walker, R. (2003). Cultural differences in emotions: A context for interpreting emotional experiences. *Behaviour Research and Therapy*, 41, 777–793.
- Mikula, G., Scherer, K. R., & Athenstaedt, U. (1998). The role of injustice in the elicitation of differential emotional reactions. *Personality and Social Psychology Bulletin*, 24, 769–783.
- Mischel, W., & Shoda, Y. (1995). A cognitive-affective system theory of personality: Reconceptualizing situations, dispositions, dynamics, and invariance in personality structure. *Psychological Review*, 102, 246–268.
- Mischel, W., & Shoda, Y. (1998). Reconciling processing dynamics and personality dispositions. *Annual Review in Psychology*, 49, 229–258.
- Mondillon, L., Niedenthal, P. M., Brauer, M., Rohmann, A., Dalle, N., & Ichida, Y. (2005). Beliefs about power and its relation to emotional experience: A comparison of Japan, France, Germany, and the United States. *Personality and Social Psychology Bulletin*, 31, 1112–1122.
- Murray, H. (1938). *Exploration in personality*. New York: Oxford University Press.
- Ohbuchi, K., Tamura, T., Quigley, B. M., Tedeschi, J. T., Madi, N., Bond, M. B., et al (2004). Anger, blame, and dimensions of perceived norm violations: Culture, gender, and relationships. *Journal of Applied Social Psychology*, 34, 1587–1603.
- Orth, U., & Wieland, E. (2006). Anger, hostility, and posttraumatic stress disorder in trauma exposed adults: A meta analysis. *Journal of Consulting and Clinical Psychology*, 4, 698–706.
- Parker, E. H., Hubbard, J. A., Ramsden, S. R., Relyea, N., Dearing, K. F., & Schimmel, K. D. (2001). Children's use and knowledge of display rules for anger following hypothetical vignettes versus following live peer interactions. *Social Development*, 10, 528–557.
- Peterson, C., Semmel, A., von Baeyer, C., Abramson, L. Y., Metalsky, G. I., & Seligman, M. E. P. (1982). The attributional style questionnaire. *Cognitive Therapy and Research*, 6, 287–299.

- Quartana, P. J., & Burns, J. W. (2007). Painful consequences of anger suppression. *Emotion*, 7, 400–414.
- Quigley, B. M., & Tedeschi, J. T. (1996). Mediating effects of blame attributions on feelings of anger. *Personality and Social Psychology Bulletin*, 22, 1280–1288.
- Rivera, C., & Wranik, T. (2008). *Effective anger management at work: Individual and contextual factors*. Paper presented at the XXIX International Congress of Psychology, Berlin, Germany.
- Roseman, I. J. (1984). Cognitive determinants of emotions: A structural theory. In P. Shaver (Ed.), *Review of personality and social psychology: Vol. 5. Emotions, relationships, and health* (pp. 11–36). Beverly Hills, CA: Sage.
- Roseman, I. J. (1991). Appraisal determinants of discrete emotions. *Cognition and Emotion*, 5, 161–200.
- Roseman, I. J. (2001). A model of appraisal in the emotion system: Integrating theory, research, and applications. In K. R. Scherer, A. Schorr, & T. Johnstone (Eds.), *Appraisal processes in emotion: Theory, methods, research* (pp. 68–91). New York: Oxford University Press.
- Rosenberg, M. (1965). *Society and the adolescent self-image*. Princeton, NJ: Princeton University Press.
- Rosenwein, B. H. (Ed.). (1998). *Anger's past: The social uses of an emotion in the middle ages*. Ithaca: Cornell University Press.
- Rotter, J. B. (1966). Generalized expectancies for internal vs. external control of reinforcement. *Psychological Monographs: General and Applied*, 80, 1–28.
- Russell, J. A. (2003). Core affect and the psychological construction of emotion. *Psychological Review*, 110, 145–172.
- Russell, J. A., & Fehr, B. (1994). Fuzzy concepts in a fuzzy hierarchy: Varieties of anger. *Journal of Personality and Social Psychology*, 67, 186–205.
- Santos, C. O., Caeiro, L., Ferro, J. M., Albuquerque, R., & Figueira, M. L. (2006). Anger, hostility and aggression in the first days of acute stroke. *European Journal of Neurology*, 13, 351–358.
- Scheier, M. F., & Carver, C. S. (1985). Optimism, coping, and health: Assessment and implications of generalized outcome expectancies. *Health Psychology*, 4, 219–247.
- Scherer, K. R. (1984). On the nature and function of emotion: A component process approach. In K. R. Scherer & P. Ekman (Eds.), *Approaches to emotion* (pp. 293–317). Hillsdale, NJ: Erlbaum.
- Scherer, K. R. (2001). Appraisal considered as a process of multi-level sequential checking. In K. R. Scherer, A. Schorr, & T. Johnstone (Eds.), *Appraisal processes in emotion: Theory, methods, research* (pp. 92–120). New York and Oxford: Oxford University Press.
- Scherer, K. R. (2004). Feelings integrate the central representation of appraisal-driven response organization in emotion. In A. Fischer, A. S. R. Manstead, & N. Frijda (Eds.), *Feelings and emotions: The Amsterdam symposium* (pp. 136–157). New York: Cambridge University Press.
- Scherer, K. R., & Ellgring, H. (2007). Multimodal expression of emotion: Affect programs or componential appraisal patterns? *Emotion*, 7, 158–171.
- Scherer, K. R., Wranik, T., Sangsue, J., Tran, V., & Scherer, U. (2004). An actuarial approach to emotions: Emotions in everyday life. *Social Science Information*, 43, 499–570.
- Schiraldi, G. R., & Kerr, M. H. (2002). *The anger management sourcebook*. New York: McGraw Hill.
- Schmitt, M. (1996). Individual differences in sensitivity to befallen injustice (SBI). *Personality and Individual Differences*, 21, 3–20.
- Schwartz, S. (1992). Universals in the content and structure of values: Theoretical advances and empirical tests in 20 countries. In M. P. Zanna (Ed.), *Advances in experimental social psychology* (Vol. 25, pp. 1–16). New York: Academic Press.
- Scott, W. D., & Steidtmann, D. (2006). Dysphoria and hostile cognition: The relationship depends on levels of trait anger. *Cognitive Therapy and Research*, 30, 19–27.
- Seligman, M. E. P. (1986). *Questionnaire de style attributionnel*. New York: Trans-Optic.
- Seligman, M. E. P. (1998). *Learned optimism*. New York: Pocket books.
- Smith, C. A., & Ellsworth, P. C. (1985). Patterns of cognitive appraisal in emotion. *Journal of Personality and Social Psychology*, 48, 813–838.
- Smith, C. A., & Lazarus, R. S. (1993). Appraisal components, core relational themes, and the emotions. *Cognition and Emotion*, 7, 233–269.
- Smith, C. A., & Pope, L. K. (1992). Appraisal and emotion: The interactional contribution of dispositional and situational factors. In M. S. Clark (Ed.), *Review of personality and social psychology: Emotions and social behavior* (pp. 32–62). Newbury Park, CA: Sage.
- Smits, D. J. M., & Kuppens, P. (2005). The relation between anger, coping with anger, and aggression, and the BIS/BAS system. *Personality and Individual Differences*, 39, 783–793.
- Spielberger, C. D. (1972). Anxiety an emotional state. In C. D. Spielberger (Ed.), *Anxiety: Current trends in theory and research* (pp. 23–49). New York: Academic Press.

- Spielberger, C., Krasner, S. S., & Solomon, E. P. (1988). The experience, expression, and control of anger. In M. P. Janisse (Ed.), *Health psychology: Individual differences and stress* (pp. 123–143). New York: Springer.
- Stemmler, G. (1997). Selective activation of traits: Boundary conditions for the activation of anger. *Personality and Individual Differences*, 22, 213–233.
- Stemmler, G. (2004). Physiological processes during emotion. In R. S. Feldman & P. Philippot (Eds.), *The regulation of emotion* (pp. 33–70). Mahwah, NJ: Lawrence Erlbaum.
- Tavris, C. (1989). *Anger: The misunderstood emotion*. New York: Simon & Schuster.
- Tomkins, S. S. (1962). *Affect/imagery/consciousness: Vol 1. The positive affects*. New York: Springer.
- Tomkins, S. S. (1963). *Affect/imagery/consciousness: Vol 2. The negative affects*. New York: Springer.
- Van Coillie, H., & Van Mechelen, I. (2006). A taxonomy of anger-related behaviors in young adults. *Motivation and Emotion*, 30, 57–74.
- Van Coillie, H., Van Mechelen, I., & Ceulemans, E. (2006). Multidimensional individual differences in anger-related behaviors. *Personality and Individual Differences*, 41, 27–38.
- Vansteelandt, K., & van Mechelen, I. (2006). Individual differences in anger and sadness: In pursuit of active situational features and psychological processes. *Journal of Personality*, 74, 871–909.
- Verona, E., & Curtin, J. J. (2006). Gender differences in the negative affective priming of aggressive behavior. *Emotion*, 6, 115–124.
- Weber, H. (2004). Explorations in the social construction of anger. *Motivation and Emotion*, 28, 197–219.
- Wehrle, T., Kaiser, S., Schmidt, S., & Scherer, K. R. (2000). Studying the dynamics of emotional expression using synthesized facial muscle movement. *Journal of Personality and Social Psychology*, 78, 105–119.
- Whalen, P. J., Shin, L. M., McInerney, S. C., Fischer, H., Wright, C. I., & Rauch, S. L. (2001). A functional MRI study of human amygdala responses to facial expressions of fear versus anger. *Emotion*, 1, 70–83.
- Wilkowski, B. M., & Robinson, M. D. (2008). The cognitive basis of trait anger and reactive aggression: An integrative analysis. *Personality and Social Psychology Review*, 12, 3–21.
- Wranik, T. (2005). *Personality under stress, who gets angry and why? Individual differences in appraisal and emotion*. Doctoral Dissertation, 336, University of Geneva, Switzerland.
- Wranik, T., Feldman Barrett, L., & Salovey, P. (2006). Intelligent emotion regulation: Is knowledge power? In Gross, J. (Ed.), *Handbook of emotion regulation* (pp. 393–407). New York: Guilford.
- Wranik, T., & Fiori, M. (2008). *Are all anger experiences the same?* Paper presented at the XXIX International Congress of Psychology, Berlin, Germany.
- Wranik, T., & Scherer, K. R. (2001). *What makes you angrier in an interactive work task: External or internal attribution?* Conference proceedings, Feelings and Emotions: The Amsterdam Symposium, Amsterdam.
- Wranik, T., & Scherer, K. R. (2009). *Anger at work: Who gets the blame?* Submitted.
- Zajonc, R. B. (1984). On primacy of affect. In K. R. Scherer & P. Ekman (Eds.), *Approaches to emotion* (pp. 259–270). Hillsdale, NJ: Erlbaum.
- Zeman, J., & Garber, J. (1996). Display rules for anger, sadness, and pain: It depends on who is watching. *Child Development*, 67, 957–973.

Chapter 16

Appraisals and Anger: How Complete Are the Usual Appraisal Accounts of Anger?

Leonard Berkowitz

In the last several years a large amount of theory and data have been produced on the role of appraisal in emotions. Both theory and data make it desirable to evaluate this role, and to provide answers to questions regarding the place of appraisal in emotion: Whether it is indispensable for emotion elicitation, and what the process of appraisal consists of. The questions are of general relevance for the theory of emotion. . .

Frijda (1993, p. 357)

Abstract Without questioning that people's appraisals of the situations they are in can greatly determine what emotions they experience, this chapter argues that traditional appraisal accounts of anger genesis are seriously incomplete and that anger can at times arise in ways not anticipated by appraisal formulations. Anger is here regarded as an experience that is part of a constellation of physiological, motoric, and cognitive responses, all related associatively to the inclination to attack and/or injure an available target. It is proposed that this feeling can arise independently of appraisals when the situation is decidedly unpleasant and/or external stimuli are present or bodily movements are made that are linked associatively with aggression. Various problems with conventional appraisal research are discussed, such as the uncertainty at times as to whether the appraisals are antecedents to or consequences of the emotion and whether the features often said to be a requirement for anger to arise are indeed necessary.

The passage of time has certainly not lessened the need recognized by Frijda (1993) for an assessment of the role of appraisal in emotion. Since Frijda made his comment, older appraisal formulations have been elaborated, new ones advanced, and scores of additional research studies have been published (see, for example, Scherer, 1999; Scherer, Schorr, & Johnstone, 2001; Chapter 15 by Wranik & Scherer, this volume). A number of writers have considered the adequacy of appraisal analyses of emotion in general, some very favorably (e.g., Roseman & Smith, 2001; Scherer, 2001b; Chapter 15 by Wranik & Scherer, this volume), and others highlight conceptual and methodological difficulties (e.g., Berkowitz & Harmon-Jones, 2004; Frijda, 1993; Parkinson, 1999; Parkinson & Manstead, 1992). The present review differs from all but the first of these in being focused on appraisal analyses of *anger in particular*.

My position regarding appraisals should be clear from the start: I have no doubt that people's evaluations of situations greatly determine what emotions they will experience. The Lerner and

L. Berkowitz (✉)
University of Wisconsin-Madison, Madison, WI 53705, USA
e-mail: lberkowi@wisc.edu

Tiedens appraisal model (Chapter 17 by Lerner et al., this volume), for example, can be helpful quite often and has considerable empirical support. Many of the phenomena at issue might conceivably be accommodated by the relatively broad-ranging newer developments in appraisal theorizing (e.g., Ellsworth & Scherer, 2003; Scherer, 2001; Smith & Kirby, 2000, 2001; Chapter 15 by Wranik & Scherer, this volume). Nonetheless, I contend that there are still a number of unsettled details to be ironed out and ambiguities to be resolved in appraisal formulations generally and, especially important, that anger at times arises in ways not anticipated by conventional appraisal conceptions. Like Leventhal and Scherer (1987) and others, I advocate a multiprocess approach to the analysis of anger arousal. Such a model in principle isn't necessarily inconsistent with current appraisal formulations. As Ellsworth and Scherer (2003, p. 585) observed, appraisal theorists "do not see their theories as incompatible with subcortical processing, autonomic responses, expressive responses, or action tendencies." However, for me at least, most appraisal-theory writings to date are seriously incomplete. Most notably, they have given insufficient *explicit* attention to how physical discomfort can elicit anger and how facial expressions and muscular movements in other parts of the body can also give rise to emotional reactions. (In my view, in Chapter 15 by Wranik and Scherer [in this volume], analysis of this latter phenomenon doesn't adequately account for much of the published research.) I argue, in sum, that anger arises sometimes in ways not anticipated by conventional appraisal conceptions. By highlighting "bottom-up" influences on anger, this chapter will show that a truly comprehensive account of anger (and perhaps other emotions) requires an explicit recognition of these effects and processes.

16.1 Guiding Conceptions and Definitions

16.1.1 Anger

Throughout this review *anger* is regarded primarily as an experience, a conscious feeling. However, in partial agreement with Averill (1982), I also conceive of this experience as part of an anger/affective aggression syndrome, a constellation of physiological, motoric, and cognitive responses, all related associatively to the inclination to attack and/or injure an available target (see Berkowitz, 1993, 1999, 2002, c.f. Chapter 25 by Spielberger, this volume). These interconnections are only partial, so that, for example, anger experiences may have only a low correlation with overt aggression. Nevertheless, the relationship is usually substantial enough to permit us to regard displays of affectively generated aggression as at least an approximate indicator of the strength of the felt anger (Chapter 14 by Hubbard et al., this volume). What is especially important is that the anger experience is linked to an urge to hurt (or even, if intense enough, to destroy) someone.¹

16.1.1.1 How Broad Should the Concept of Anger Be?

The conception of anger favored here is much broader than is proposed in some appraisal models. Clore, Ortony, Dienes, and Fujita (1993) and others have argued that many cases of aversively-triggered affective aggression are not really *emotional* reactions. They argue, "The term *emotion*

¹In emphasizing this associated impulse to injure, I differ from other writers, such as Chapter 18 by Schultz and his colleagues and Chapter 15 by Wranik and Scherer (both papers in this volume), who assume that anger is only linked to an urge to change the anger-eliciting situation or is merely a form of self-assertion.

[italics in original] is reserved for instances in which the characteristic physiology, feelings, and behavior of emotion is a reaction to an appraisal or evaluation rather than arising from other causes. . . . cognitive appraisals are a necessary condition. . . . emotion does not refer to physiology, feelings, or behaviors per se, but to physiology, feelings, and behaviors that are triggered by appropriate cognitive analyses" (p. 62). Thus, Clore et al. (1993) maintained that people may have anger-like feelings in response to certain (e.g., painful) stimuli, but that these reactions do not constitute true anger unless the requisite appraisals occur.

One way to understand the Clore et al. (1993) exclusion of "anger-like feelings" from the realm of supposedly "true" anger is to say that there are different kinds of anger. The Scherer (1999) and Chapter 15 by Wranik and Scherer (this volume) reference to a "reflexive pain reaction", also seems to suggest that pain-elicited anger is somehow different from, say, the anger produced by an intentional affront." Scherer's (1993, 2001a) distinction between "irritation/cold anger" and "rage/hot anger" is an even more explicit argument for types of anger.² Scherer (2001a) also seems to distinguish different types of anger. He proposes one pattern of appraisals for "irritation/cold anger" and another pattern for "rage/hot anger" (p.115, see also Wranik & Scherer, this book.)

In accord with the prototype view of emotion (Shaver, Schwartz, Kirson, & O'Connor, 1987; Russell & Fehr, 1994), my own conception of a very general anger embraces all the "nuances" just mentioned. The classical approach to language concepts followed by appraisal theorists regards terms such as *anger* as "...defined by a set of common features, each necessary and together sufficient" (Russell & Fehr, 1994, p. 186). By contrast, prototype-based formulations recognize the ample evidence (e.g., Shaver et al., 1987) that the general experience of anger includes a wide variety of feelings that can be called, among other things, *irritation* or *annoyance* or *exasperation* or *disgust*, as well as *anger*, and are not clearly differentiated.³ Supporters of the prototype perspective (e.g., Shaver et al., 1987) have shown that in ordinary life people do not necessarily experience anger in ways that meet the criteria of appraisal analyses, such as a perceived obstacle to a desired goal and/or an appraised injustice.

16.1.2 Appraisals

There is considerable, but not complete, agreement on the theoretical status of appraisal theory and the major components of the construals associated with various emotional states (see, for example, Scherer, 1999, p. 639; Ellsworth & Scherer, 2002. Oatley & Jenkins, 1996, also summarize several appraisal analyses.) However, there are surprising differences among several of the best known formulations as to just what *appraisals* are, and ambiguities persist and questions remain about how they function.

16.1.2.1 Are Appraisals Antecedents of Emotions?

For Ellsworth and Scherer (2002), this term has to do with features of the emotional experience, such as its valence, how much attention it commands, the perceived level of uncertainty as to what

² I suggest that these supposed differences are primarily due to differences in intensity.

³ Recognizing this substantial overlap, Ortony et al. (1988) did not distinguish among the "many nuances," and listed *anger*, *annoyance*, *exasperation*, *fury*, *incensed*, *indignation*, *irritation*..." and several other similar feelings as "tokens" of anger.

is happening, and even one's ability to cope with the event. But more commonly, construals are regarded as antecedents and causes of emotional states. As Examples, Roseman, Spindel, and Jose (1990, p. 899) say, in accord with Clore et al. (1993), that "... evaluations and interpretations of events, rather than the events per se, *determine* [emphasis added] whether an emotion will be felt and which emotion it will be". Ellsworth (2002, Personal Communication) seeks to reconcile these two views by proposing that when people first become emotional, their experience is initially appraised at some minimum level of awareness along the dimensions she and her colleagues have identified. Then, "when enough appraisals that are diagnostic of a named emotion exist" (Ellsworth, 2002, personal communication), the particular emotion is consciously felt. In her view these latter appraisals *cause* the experienced emotion in the same sense that the color *green* is produced by the colors *blue* and *yellow* (also see Ellsworth & Scherer, 2002, for a further explication of this perspective).

Another important ambiguity in the theorizing about appraisals stems from the verbal reports used in most investigations. As Frijda (1993; Frijda & Zeelenberg, 2001) and Parkinson and Manstead (1992) have noted, it is often impossible to ascertain whether the identified appraisal characteristics preceded or followed the emotional experience. "Nothing in the data [obtained in these studies] resists the interpretation that the relevant appraisals were consequences rather than precedents of the emotional reactions" (Parkinson & Manstead, 1992, p. 129).⁴ Appraisals as antecedents or consequences is especially pertinent to analyses of anger; and we will return to this question.

16.2 Appraisal Features Theoretically Responsible for Anger

Let's now look into the adequacy of several of the causal propositions appraisal theorists have advanced to account for anger arousal. Some of the main ideas regarding these supposed anger determinants are summarized in Table 16.1 (cf. Stein & Levine, 1990, 1999 for alternative formulations.) Although appraisal theorists typically trace any given emotion to a pattern of appraisals rather than to any one construal component alone (Roseman & Smith, 2001, p. 16),⁵ this Discussion will take up each component in turn for simplicity's sake.

16.2.1 Goal Attainment

Many appraisal theories start by assuming that people frequently are concerned about reaching their particular goals. As Scherer (2001a, p. 96; also see Chapter 15 by Wranik & Scherer, this volume) puts it, "The central tenet of appraisal theory is that it is not the event itself but the perceived outcomes for the individual [i.e., what the person believes are the situation's implications for her or his goals in that situation] that determines the ensuing emotion." Unsurprisingly, several analyses hold that, if there is to be an angry reaction, the instigating occurrence must be understood as personally significant in some way, as having a *goal* or *motivational relevance* (e.g., Lazarus, 1991,

⁴Robinson and Clore (2001) have defended the frequent reliance on vignette- and recall-based methods in emotion investigations by reporting research indicating that appraisal-emotion relations uncovered by these methods can be similar to the appraisal-emotion relations identified by a direct emotion-elicitation procedure. However, as they acknowledged, the issue is by no means settled and further research is necessary.

⁵Adopting the characterization employed by Ellsworth and Scherer (2003), we can say that we are here dealing with the "profile" of features a number of appraisal theorists consider to be "necessary and sufficient" for the construction of the anger experience.

Table 16.1 Appraisal features theoretically associated with anger

	Lazarus (1991)	Ortony et al. (1998)	Roseman (1991)	Scherer (2001a)
Motivational relevance	X			X
Frustration	X	X	X	X
External cause	X	X	X	X
Other blame	X	X		
Improper		X	X ¹	X
Coping potential	X	X		X

¹Roseman's (1991) conception of *impropriety* (or *illegitimacy*) is somewhat different from Scherer's (2001a), and moreover, his view regarding the nature and effect of this appraisal component changed in later papers (e.g., Roseman et al., 1996). This is discussed in the text.

I have not included Weiner (e.g., Weiner et al., 1982) in this table only for reasons of limited space. With the others cited, he too sought to identify the characteristics of a situational interpretation that determines whether anger will arise (Strictly speaking, though, his formulation is often labeled *attribution theory* rather than *appraisal theory* [e.g., Smith et al., 1993]).

p. 222; Smith & Lazarus, 1993, p. 237; Scherer, 2001a, p. 95; also see Frijda, 1993; Smith, Haynes, Lazarus, and Pope, 1993, p. 916). However, accounts differ as to how important the goals must be to generate anger. Roseman (1991) refers only to whether the situation is appraised as related to a reward the persons desire or a punishment they hope to avoid. Scherer (2001a, p.95) stresses that it is interference with "major goals/needs" that is the key, while Lazarus (1991, p. 222) argues that a particular "... *type of ego-involvement* [emphasis in original], must ... be activated for anger to occur". All these theorists propose that anger-instigating events are basically unpleasant, mainly because the satisfaction of some objective is blocked. Two formulations (Ortony, Clore, & Collins, 1988; Weiner, Graham, & Chandler, 1982) more or less take this as a given. Roseman (1991) traces anger somewhat more specifically to the absence of a reward or the presence of punishment; for Lazarus and associates (e.g., Lazarus, 1991; Smith & Lazarus, 1993) and Scherer (2001a, p. 96) the appraisal must identify an obstruction to (or a more general incompatibility with) the satisfaction of some goal. The idea that an impediment to goal attainment is at least contributory to anger arousal obviously has some similarity to the classic frustration-aggression thesis (Dollard, Doob, Miller, Mowrer, and Sears, 1939), but this discussion is beyond the scope of this chapter.

16.2.2 External Agency and Blame

All these writers (and many others [e.g., Ellsworth & Smith, 1988; Roseman, Antoniou, & Jose, 1996; Scherer, 1993; Smith & Ellsworth, 1985]) argue that someone or some thing, an external agent, must be seen as responsible for the negative event if there is to be anger. Indeed, some studies (e.g., Ellsworth & Smith, 1988) found that the perception that an external agent had brought about the unpleasant event was a principal determinant. Weiner (e.g., Weiner, Graham, & Chandler, 1982) adds another feature to this notion of an external causal agent: that this other-agent had the power to control what had happened. (It is unclear whether Scherer, 1999, 2001a, also believes in the importance of the external agent's perceived power over what happens; his conception of the power to control an occurrence has more to do with the appraiser's *own* power rather than that of the other-agent (also see Ellsworth & Scherer, 2002)).

Even though all of these theorists postulated the necessity of an external entity being regarded as the cause of the impediment (or more generally, the unpleasant occurrence), only two (Lazarus, 1991; Ortony, Clore, and Collins, 1988) explicitly state that someone or some thing must be *blamed* for the negative event if anger is to arise. For Lazarus (1991, 2001) and associates (e.g., Smith & Lazarus, 1993) appraisals can be understood in terms of their specific details or, at a more molar

level, their “core relational theme,” the details taken together form a general conception. The Lazarus group unsurprisingly said the overall theme involved in anger generation is “other blame.”⁶ But even though this kind of general interpretation can play an important part in anger production, the more molecular level appraisal components might also make a separate contribution to this emotion. Smith and Lazarus (1993) found that measures of core relational themes and also of the separate appraisal components were related to self-reported anger. The authors concluded that these “two levels of analysis are complementary” (p. 259).

16.2.3 Legitimacy

There is an interesting divergence of views regarding the perception of the instigating incident as unfair or improper. Surveys of ordinary persons’ conception of their angering experiences (e.g., Averill, 1982; Shaver et al., 1987), as well as appraisal theory-guided investigations (e.g., Frijda, Kuipers, & ter Schure, 1989), have found that the provoking event is often regarded as illegitimate, “contrary to what ought to be” (Shaver et al., 1987, p. 1077). Ortony et al. (1988) seemed to have this general idea in speaking of the event’s blameworthiness. Writing on the philosophy of emotion, Solomon (1993), insisted that there cannot be anger unless there is a perceived offense. In his words, “if there is nothing objectionable... or offensive (to the person), then those feelings do not count as... anger (or even as ‘feeling angry’)...” (p.10).⁷ For Roseman (1991), the obtained outcome was illegitimate when the person believed she/he had deserved results that could not be obtained or had not deserved the punishment that was received. Supporting these views, Weiss, Suckow, and Cropanzano (1999) experimentally manipulated the unfairness of the outcome received by their participants, and showed that the injustice produced an angry reaction.

However, even with this support, appraisal research has uncovered some problems. In some studies (e.g., Ellsworth & Smith, 1988; Mikula, Scherer, & Athenstaedt, 1998) the participants’ judgment of the illegitimacy (or unfairness or injustice) of the instigating event was strongly associated with other perceived features of the event, so that it is unclear *in these investigations* how much, if any, of the variance in the reported anger was due to the incident’s appraised impropriety. Because of ambiguities such as these as well as some non-replications, Roseman et al. (1996) suggest (1) that illegitimacy may be a typical – but not a necessary – determinant of anger and (2) that this feature affects anger through the operation of another appraisal component – control potential. For Roseman et al. (1996), people believing they deserved a happier result have a sense of themselves as powerful and having some control over what can happen. This appraisal of control potential presumably enables their anger.

As Table 16.1 indicates, other appraisal theorists share the Roseman et al. (1996) contention that a perceived ability to control the instigating event is necessary if anger is to arise, although there are differences in detail (also see Roseman et al., 1996, pp. 246–247). Roseman and associates (e.g., Roseman et al., 1996, p. 262) seem to define this coping generally as the ability to do something about the event’s “motive-inconsistent aspects.” Scherer (e.g., 1993, 2001a) also

⁶Lazarus’ (1991) version of the core relational theme actually is stronger. Believing that the offense has to be taken as an attack on the self if anger is to arise, he maintains that anger is evoked when one construes the event as a “demeaning offense against me and mine” (p. 222).

⁷Solomon here was following the classical approach to language concepts in which a concept “is defined by a set of common features, each necessary and together sufficient to determine membership” (Russell & Fehr, 1994, p. 186). We favor the very different prototype perspective.

viewed an appraised coping potential as the belief that one has the ability (or power) to control the “consequences.” Stein and Levine (1989, 1990, 1999) regard the coping belief differentiating anger from sadness as involving the perceived ability to remove the frustrating obstruction and attain the desired goal. However, no one specified whether frustration was to be overcome through problem solving, aggression, or both. I suggest below that *something like* this factor does indeed play an important part in determining the likelihood of an anger response to a stressful event, but perhaps not in the way the appraisal theorists propose.

16.3 Research into Conditions Generating Anger

I now review a sampling of studies bearing on the foregoing propositions, noting some uncertainties and qualifying or broadening some of these ideas. Contrary to the often-stated claim that the appraisal features just covered are both necessary and sufficient (in some combination) for anger generation, I will show that these appraisal components can facilitate anger arousal, or affect its intensity, but are not necessary. To paraphrase Roseman et al. (1996), these appraisal features may be typical of many angering experiences without being required for anger to occur. Note also that some of the factors often identified as contributors to anger arousal may actually have little role in what most people regard as their typical angering experience. As one case in point, about 14% of the anger-arousing events reported in Averill’s (1982) survey of community residents and university students were not in keeping with conventional appraisal theory. A respondent in Russell and Fehr’s (1994, p. 194) study reported becoming angry as the result of a mild accident. Such accidents may not be a major source of everyday anger (this participant regarded the event as “only a slightly good example” of anger-producing incidents), but painful accidents can indeed create bursts of anger (Frijda 1993.) A truly comprehensive theory of affective states should attempt to deal with relatively unusual occurrences as well as the more common ones.⁸ (Actually, we cannot be sure that pain-elicited anger is so infrequent. It might occur more often than study participants recall or guess; many people may be reluctant to admit being angered by a painful or otherwise socially improper event because they think that such anger is unreasonable [see Parkinson, 1999].)

16.3.1 *Must the Frustration Be Personally Significant?*

As noted earlier, several appraisal analyses hold that people have to be seeking a personally significant objective if an impediment to its achievement is to be angering (e.g., Lazarus, 1991; Scherer, 2001a, 1984; Chapter 15 by Wranik & Scherer, this volume). However, several experiments have demonstrated that the failure to obtain an expected gratification can generate an aggressive inclination even when the failure is not a blow to self-esteem. In one of these (Walters & Brown, 1963) youngsters who were unable to watch a promised enjoyable film because the movie projector had “accidentally” broken down were especially aggressive to a peer during a subsequent game.

Research with human infants also indicates that frustrations can give rise to aggressive inclinations even in the absence of earlier training to be aggressive. Employing an experimental

⁸We should note that Festinger (1957, see Harmon-Jones & Mills, 1999) developed his theory of cognitive dissonance, a theory relevant to a good deal of everyday thinking and behavior, in part on the basis of rare events – the prediction by a cult that the continent would be destroyed and also the spreading of rumors after catastrophes.

procedure fairly close to the appraisal theory conception of frustration, Lewis (1993, also this book) conditioned 2- to 8-month-old infants to move an arm to see a picture of a baby's smiling face. Few anger-like facial expressions were exhibited during this training. By contrast, in the subsequent extinction phase, where the arm movement no longer revealed the pleasant picture, the great majority of the infants showed the anger-like facial expressions.⁹ Lewis (1993, also this book) held that this frustration reaction was "built-in."

16.3.2 Must the Frustration Be Improper?

Some, but not all, appraisal proponents argue that anger is a response to a perceived offense (e.g., Solomon, 1993) or, put another way, to an external agent's blameworthy behavior (Ortony et al., 1988; Clore et al., 1993).¹⁰ Despite its great popularity, a considerable body of evidence indicates that this contention should be qualified.¹¹

There is no doubt that anger often results when another person is seen as acting "wrongly" (e.g., in addition to appraisal-based studies, see Baron, Neuman, & Geddes, 1999, and Weiss et al., 1999). But it also appears that the ordinary "person in the street" (to use Averill's, 1982, characterization) does become angry even when there is no such construal. Averill (1982) found that the majority of angering incidents (59%) reportedly followed "a voluntary and unjustified act," approximately 12% were produced by "a voluntary and justified act," and another 2% by "an unavoidable accident or event" (Averill, 1982.) Similarly, Russell and Fehr (1994, p. 194) quoted reports of instances in which anger was not caused by an external agent's supposedly improper behavior.

Laboratory experiments also indicate that anger and/or affective aggression can be generated by negative events that presumably are not appraised as improper. Participants in the Dill and Anderson (1995) study faced a difficulty in working on their assigned task; the experimenter provided a reasonable justification for some groups, and an arbitrary, unjustifiable explanation for others. Unsurprisingly, participants, given the unjustifiable impediment, expressed the greatest hostility when rating the experimenter later. However, even the people, given the justifiable difficulty, were more hostile than the nonfrustrated control group. The presumably proper barrier to goal attainment apparently was unpleasant enough to elicit some hostile inclinations (also see Geen, 1968, for relevant evidence.)

⁹Appraisal theorists do not agree in their interpretations of findings such as these. Clore et al. (1993) maintained that anger-like facial expressions are not in themselves indicators of anger emotion. They said "one can have an emotional expression, engage in emotional behavior. . . [but] these constitute emotions only when they are reactions to the cognitive representation of something as good or bad" (p. 63). Stein and Levine (1999), on the other hand, were willing to accept babies' facial expressions as signs of a true emotion since the facial movements indicate that the babies had representations of a changed goal state (p. 387).

¹⁰On the basis of their analysis of the reported anger experiences of almost 3,000 students in 37 countries, Mikula et al. (1998) concluded that perceived injustice was present to some extent in many negative emotions, and that other appraisal components (such as the perceived causal agent and coping ability) also contributed to the differentiation of anger from these other negative states.

¹¹We pointed out earlier that Roseman et al. (1996) questioned the idea that the instigating event has to be viewed as illegitimate (or blameworthy or a violation of one's standards) if anger is to arise. They gave the findings obtained by Stenberg and his colleagues (e.g., Stenberg, Campos, & Emde (1983)) as one reason for their position: "infants appear to experience anger. . . before they would seem to have the cognitive capacity to make sophisticated judgments of legitimacy" (p. 271).

16.3.3 Blame: The Problem of Causal Direction

I noted earlier that many appraisal proponents essentially agree with Averill's (1982) contention that anger is an accusation of blame. However widely shared, this view faces an important difficulty: We cannot unequivocally determine the causal direction of the connection between anger and blame in many of the relevant studies. The blaming may have been, in some cases at least, an "epiphenomenon," a consequent rather than an antecedent of anger arousal (Frijda, 1993). Quigley and Tedeschi's (1996) LISREL analysis of people's descriptions of anger-arousing events supported such a possibility. They found that the data could reasonably be accommodated by a model in which "anger and blame exist in a reciprocal relationship" (p. 1280). Stein and Levine (1989, 1990, 1999) essentially raised this possibility in suggesting that people blame an external agent when they are emotionally upset, at least in part because they have learned the benefits of doing so. Their observations suggested that pre-school-age children have not yet learned to blame others when angered by a frustration.

Frijda's (1993) "top down" explanation of why anger can elicit blame appraisals was that angry feelings can prompt a person to seek someone who can be held responsible for the provoking aversive situation. Bower's (1981) associative network account of emotional effects on thoughts and memory suggests a "bottom-up" explanation of how angry people could become disposed to blame others. If we have frequently been angered by other persons' actions, an associative link will develop in our memory between the "node" representing our anger state and the idea that someone else is responsible for the upsetting event.¹² Once this connection forms, even an anger-arousing occurrence that cannot be blamed on another could activate thoughts of other people's responsibility. Keltner, Ellsworth, and Edwards (1993) have shown just this. Undergraduates in one of their experiments were first induced to become either angry or sad by requiring them to adopt the physical pose characteristic of these emotional states, and then were asked to rate the causes of important circumstances in their lives. In contrast to their sad counterparts, the angry participants typically were more likely to attribute their life circumstances, as well as the problems they thought they might encounter in the future, to other people's actions. The mere performance of anger-associated skeletal-motor movements had led to angry feelings which evidently then activated ideas often associated with this affective state, such as "others made me feel this way." (Also see Lerner & Keltner, 2000, 2001; Siemer, 2001.)

16.3.4 Must There Be an External Cause of the Negative Event?

Despite the agreement among appraisal theorists that external agents are typically viewed as being responsible for the anger-producing event, both clinical observations and experimental results indicate that decidedly aversive conditions can generate anger even when they are not caused by an outside entity. We can see this at times in people suffering from chronic pain. According to quite a few studies (see, e.g., Hatch et al., 1992; Venable, Carlson, & Wilson, 2001), people afflicted by recurrent headaches are not infrequently described as often angry and/or hostile.

Of course, in at least some of these cases the anger might contribute to the headaches. Nonetheless, laboratory experiments in which physical discomfort or pain is deliberately established

¹²The linkage between anger state and other accountability can be bidirectional. Neumann (2000) demonstrated that a heightened readiness to think of other persons as active causal agents increases the likelihood of having angry feelings in response to negative events.

have shown that the decidedly unpleasant physical sensations in themselves can be angering and lead to affective aggression (see Berkowitz, 1993; 1998; 1999, 2002). What is perhaps even more intriguing, psychological discomfort, that is not physically painful, can also arouse anger, even when the discomfort cannot be attributed to an external cause. In Mikulincer's (1988) study, as one example, some of his participants were made to fail only moderately in the task given them so that they did not become completely apathetic to working on their assignment. Not having given up entirely, these persons reported feeling angry and frustrated. What is important here is that it was the individuals who attributed their failure to *internal* factors who reported feeling the most anger. They did not have to fault an external source in order to be angry. Geen (1968) has provided even more direct evidence. In his experiment the participants worked on a jigsaw puzzle in the presence of a supposed "other student." In one condition the people were thwarted in their efforts because of the distractions created by this other person, whereas for other participants the puzzle (unknown to them) actually was insoluble so that it seemed they themselves were responsible for their failure. When all of them were later given an opportunity to administer electric shocks to the "other student," those who had been frustrated by this individual were most punitive to him. But even the people who believed their failure was internally caused were more aggressive to their peer than were the nonfrustrated controls.¹³

16.3.5 *Some Questions About Coping Potential*

I now come to the challenging proposition favored by some, but not all, appraisal theorists: that people will not become angry unless they believe they can cope with the provocative occurrence — that they must see themselves as having *coping potential*. I wonder whether angry persons always think, consciously or unconsciously, that they can master the disturbance even a fraction of a second *before* this emotion arises. Angry outbursts are at times very impulsive, and not a few of those who had been carried away by their strong anger have later bemoaned their loss of control (see Potegal, this book). Even ordinarily well-behaved university students can at times display this impulsivity. According to Shaver et al. (1987), the students' anger prototype tends to include such features as "Loud voice, yelling, screaming, shouting," "Attacking something other than the cause of anger," and even "Incoherent, out-of-control, highly emotional behavior" (p. 1078). Similarly, a number of the students who were queried about their anger experiences by Roseman, Wiest, and Swartz (1994) reported that they had "felt blood rushing through the body and felt as if they would explode... and felt like yelling and like hitting someone" (pp. 212–213). Assuming these and similar statements are veridical, we wonder if these apparently involuntary urges indeed grow out of the angry persons' appraisal, even a nonconscious one, that they can resolve the disturbing situation. Do they not seem more like a strong impulse to strike at the aversive target rather than the product of some calculation that a difficulty can be overcome?

Perhaps more to the point, the only empirical support for the coping potential proposition is correlational in nature: In the appraisal studies supposedly showing that angry persons think they

¹³Following Gigerenzer (1991), it may be that psychologists' adherence to R.A. Fisher's approach to significance testing, with its emphasis on avoiding Type I errors and neglect of the possibility of Type II errors, makes it easy for us to conclude that a particular condition (e.g., a legitimate frustration) is *not at all* an influence on the dependent variable (e.g., rated anger) — rather than a weak influence — unless this condition led to a result that is statistically significant at the traditional .05 level. Furthermore, since the probability of a significant effect is largely determined by sample size, an effect may have existed in a number of published studies that was not revealed through significance testing because of a small sample (or other factors contributing to low statistical power).

can overcome the difficulty facing them, the reported sense of control did not clearly precede the felt anger and only accompanied this emotion.¹⁴

Moreover, as Chapter 5 by Harmon-Jones and his associates (this volume) report, several laboratory experiments indicate that anger can occur even when those afflicted by the negative event cannot successfully deal with the unpleasant occurrence. Harmon-Jones, Sigelman, Bohlig, and Harmon-Jones (2003), directly tested the effects of perceived coping ability, defined generally as the possibility of eliminating a moderate threat. The student participants, who were all opposed to tuition increases, first were informed either that such a raise in tuition definitely would occur or, in other cases, that the increase was still not definite and that petitions were being circulated in opposition to the jump in tuition costs. All of the students then listened to an angering "radio editorial" arguing for increased tuition. As the investigators had predicted, it was only when the students had been led to believe it was possible to fend off the bothersome tuition increase that there was a significant increase in a particular form of brain activity indicating a readiness to take action (in this case by signing the supposed anti-threat petition). These people, we can say, perceived a high coping potential. But, contrary to the appraisal theory-coping proposition, the participants' later self-reported anger was just as high when action was not possible (and supposedly nothing could be done about the disturbance) as when the possibility of eliminating the threat existed. In this experiment, then, coping potential did not affect the level of self-reported anger.

Other experimental findings also suggest that people can become angry even when they do not think they can master the problem facing them (Geen, 1968; Mikulincer, 1988). Interestingly, experiments concerned with the effects of learned helplessness also point in this direction. In their review of the learned helplessness research up to the late 1970s, Miller and Norman (1979, p. 96) reported that as the participants learned to feel helpless in the situation before them, many of them became hostile. The growing sense of being unable to master the difficulty facing them evidently promoted anger.¹⁵

Given these experimental results, the correlational nature of the findings obtained in the appraisal studies of coping potential is clearly problematic. It is by no means definite that a sense of being able to overcome the confronting disturbance is necessary for anger arousal. However, other reactions, also at times found to be correlated with coping potential appraisals, suggest why this perception is at times related to anger generation. In several of these appraisal investigations angry persons not only believed they had the power to master the difficulty confronting them, but also felt they became "stronger (higher in potency) and more energized in order to fight or rail against the cause of anger" (Shaver et al., 1987, p. 1078). Angry people are sometimes also apt to think they had relatively high control over events, are optimistic about their lives, and are willing to make relatively risky decisions (Lerner & Keltner, 2001). This sense of potency/control might be the phenomenal counterpart of the particular brain activity recorded by Harmon-Jones et al. (2003). In other words, the coping potential appraisal as well as this sense of being energized and powerful might parallel rather than cause the anger arousal. It could well reflect the physiological, motoric, and cognitive reactions to an anger-inducing event.

¹⁴Even correlational studies have not always found that the sense of being able to overcome the difficulty is related to anger. Thus, contrary to Roseman's theoretical expectation, the Roseman et al. (1990) data "revealed that people did not perceive themselves as particularly powerful in situations leading to frustration, anger, and regret" (p. 911).

¹⁵These observations, it should be noted, are not necessarily inconsistent with Seligman's (1975) formulation of learned helplessness. Seligman's conception holds that learned helplessness leads to apathy and an unwillingness to engage in the deliberate, effortful pursuit of a goal. But this does not mean that the persons feeling helpless will not experience anger or not display impulsive acts of aggression.

16.4 Other Influences on Anger Arousal

16.4.1 Aversive States of Affairs

Appraisal theorists have given relatively little attention to the question of just *why* the appraisal components postulated to generate anger have this effect. For me (Berkowitz, 1983, 1989, 1993), however, the basic reason is that many of these interpretations affect the unpleasantness of the given situation. The terms *frustration*, *goal incongruence*, *obstacles to goal attainment*, *negative outcomes* all refer to an aversive condition, a state of affairs the person ordinarily seeks to escape or avoid.¹⁶ It may well be, then, as my reformulation of the frustration-aggression hypothesis holds (Berkowitz, 1989), a barrier to the achievement of an expected gratification activates the anger/affective aggression syndrome only to the extent that intense displeasure is experienced. And by the same token, intentionally inflicted harm, unjustified thwartings, and ego threats are usually more unpleasant than accidental injuries, socially proper frustrations or impediments to the attainment of personally insignificant objectives, and the former therefore are apt to generate stronger anger and affective aggression.

Other writers (e.g., Lindsay & Anderson, 2000), even in the appraisal theory camp (Stein & Levine, 1989, 1990, 1999), have also recognized the anger/aggression-eliciting effects of aversive conditions. In summarizing one of their studies Stein and Levine (1990) concluded that their results were in accord with Berkowitz's (1983, 1989) thesis "that aversive events. . . prime anger, irritation, and hostility across a variety of contexts" (p. 69). Before proceeding further, however, it is important to point out that Berkowitz's analysis (1983, 1990, 1993, 2002; also see Geen, 1998), labeled a cognitive-neoassociationistic (CNA) model, does not claim that anger will always be seen after a decidedly unpleasant occurrence or that other factors cannot intervene to determine what emotions are manifested.

CNA is a multistage, multiprocess formulation proposing that there can be different reactions to the decidedly unpleasant occurrence depending upon what processes are in operation. Simply put, CNA suggests that initially, right after an aversive event is encountered but before cognitive control processes go into operation, the resulting negative affect automatically activates feelings, thoughts and memories, and motor impulses, all associatively linked together in an anger/affective aggression syndrome. Other syndromes such as one associated with the fear/flight pattern might also be evoked at the same time. A host of factors – genetic, learned, and situational – govern the relative dominance of these different emotional constellations, but according to the model, several syndromes can be activated at the same time, although to different degrees.

For CNA, then, it is not fight *or* flight; in some instances at least, and to some extent, both inclinations can occur together. Although there unfortunately is not much direct evidence of this at the human level, observations suggestive of the co-occurrence of fear/anxiety and anger can be found in several studies of people facing the threat of death. In one such investigation (Sugimoto & Oltjenbruns, 2001), police officers exposed to death-related stressors and showing symptoms of post-traumatic stress disorder at times exhibited inappropriate outbursts of anger, and in another (Toren, Wolmer, Weizman, Magal-Vardi, and Laor, 2002), Israeli citizens traumatized by real and then threatened missile attacks reported feeling angry as well as highly anxious. Perhaps more to the point, Miller's (1948) internal-conflict model of hostility displacement implicitly posits the co-existence of fear/anxiety-based "avoidance" tendencies and anger-derived "approach" inclinations.

¹⁶Similarly, the three types of offenses singled out by Tripp and Bies in their chapter in this book – goal obstructions, rule violations, and status/power derogations – are also aversive occurrences.

Very much in accord with Miller's thesis, my reanalysis of Fitz's results (1976, cited in Berkowitz, 1998, pp. 54–55) demonstrated that the fear arousal established in angry people led to very little aggression toward the tormentor, but still, the frightened-angered persons assaulted another individual identified as the tormentor's friend much more strongly because it was safe to do so. In this case at least, as the Miller (1948) analysis and our model both propose, anger was not eliminated by a fear-arousing threat.

CNA then maintains that with more elaborated, "higher order" cognitive processing, interpretive schemes, social rules, and anticipated costs and benefits can come into play so that the initial affective and action tendencies can be altered. If the first stage reactions are not too strong, it is presumably at this later time that appraisals can have a primary role in shaping what the person will feel and do.¹⁷

16.4.1.1 Pain and Stress

Physical pain is a clear Example of an aversive state of affairs, and as was indicated earlier, a rapidly accumulating body of research shows that pain often produces anger. This can be seen, for example, in the high levels of anger often observed in people experiencing chronic pain for any of a wide variety of reasons including arthritis, severe episodic headaches, and spinal injuries (Fernandez & Turk, 1995; Hatch et al., 1992). The anger is not always revealed openly, of course, but even so, can at times be detected by subtle, indirect measurements and/or by "anger-in" assessments of "bottled-up" anger (see Fernandez & Turk, 1995, p. 169). Correspondingly, conditions that ameliorate physical discomfort can lessen the anger produced by the aversive experience. As just one illustration of this, Weber and colleagues (2002) demonstrated that relaxation training reducing the stress of the persistent ear-ringing of tinnitus also lowered the anger generated by this disturbance.

In their discussion, Fernandez and Turk (1995) properly pointed out that one cannot exclude the possibility that appraisals played at least some part in the pain-anger relationship. However, laboratory experiments, such as Anderson's investigations of the anger-producing consequences of unpleasantly hot and cold temperatures (Anderson & Anderson, 1998) and the studies of people exposed to the cold pressor procedure reported by Berkowitz, Cochran, and Embree (1981), indicate that persons facing physically uncomfortable conditions can become angry and hostile even when it is unlikely that they made several of the construals identified in appraisal theorizing as the presumed anger-evoking profile. As one case in point, Zillmann, Baron, and Tamborini (1981) found that people exposed to unpleasant secondary cigarette smoke were relatively hostile to a nearby individual even when this person was clearly not responsible for the aversive state of affairs (also see Berkowitz, 2002).

The research on the beneficial consequences of the perceived ability to control aversive stimulation adds to the doubts we previously expressed about the presumed anger-eliciting effect of an appraised control potential. In an experiment by Geen (1978), the participants who believed they could eliminate a very unpleasant noise to which they were exposed were less punitive to a person who had provoked them earlier than were their also-provoked counterparts who lacked this perceived control over the aversive noise. Comparable results have been reported by Donnerstein and Wilson (1976) in a similar experiment.

Social and economic stress. Being decidedly unpleasant, social stresses can also generate anger and aggressive inclinations. Some of the best known Examples of the affective aggression-inducing

¹⁷Scherer's (e.g., 1993, 1999) conception of an invariant sequence of "stimulus evaluation checks" also posits a multistage appraisal process. However, where CNA suggests that the later stages come into operation only if additional thought is given to the situation at hand, the Scherer model seems to maintain that the sequence unfolds more or less automatically if the preceding "check" is satisfactory.

effects of stressful conditions can be seen in studies dealing with the consequences of economic hardships. Essentially supporting the findings originally reported in 1940 by Hovland and Sears and then corroborated by a more sophisticated reanalysis of the data conducted by Hepworth and West (1988, cited in Berkowitz, 2002), Green, Glaser, and Rich (1998) reported a significant relationship between sudden drops in the market value of cotton in the U.S. south and the lynching of African-Americans in that part of the country, but only for the period up to the Great Depression and not afterward. Evidently, whatever aggression inclinations arose from the region's economic troubles were displaced onto African-Americans in this exceedingly violent fashion only when widespread cultural attitudes and values in the South defined such people as dangerous and also permitted these kinds of assaults. Yet other research also indicates that economic frustrations can breed aggression. Studying a representative panel of people with no history of violent behavior, Catalano and colleagues (1993) found that those persons who were laid off from work after the first interviews were later much more likely to report serious acts of aggression than were their counterparts who remained employed. This significant effect survived controls for a variety of demographic measures including psychiatric history and alcohol disorders.

16.5 Effects of Emotion-Related Muscular Movements

The appraisal literature has largely ignored, or at most questioned (Tourangeau and Ellsworth, 1979) the many studies showing that facial and bodily actions can affect emotional experience (see, e.g., Tomkins, 1962, 1963; Izard, 1971; Ekman (1984, 1993); and Laird (e.g., Laird, 1984; Laird & Bresler, 1992)).¹⁸ In highlighting this extensive research area to encourage its greater integration into appraisal theory, I will focus primarily on a few of those experiments dealing with the arousal or intensification of anger.

Adelmann and Zajonc (1989) distinguished between those studies in which the bodily movements (a) modulated existing feelings, either intensifying or weakening an already established experience and (b) those in which the muscular movements initiated the emotional state. In one of the relatively few investigations showing a modulation influence on angry feelings, Jo's (1993) participants adopted either an anger-like or sad-like posture as they talked about an earlier anger- or sadness-arousing or neutral event. The anger-associated posture intensified the anger generated by the recollection of the angering incident, and furthermore, had no effect on self-reported sadness, anxiety, or cheerfulness.

Many more experiments indicate emotion-initiating effects. In two experiments employing the same postures that Jo used, Duclos, Laird, and colleagues (1989) investigated, first, the influence of sad, fear, disgust, and anger facial expressions, and second, the consequences of fear, sad, and anger body postures. In both experiments the muscular movements typical of the given emotional state led to the highest level of the particular feeling characteristically accompanying that form of expression. In another investigation when the facial expression linked to a particular emotion matched the posture adopted, the combination of these two types of movements produced the strongest emotional feelings (Flack, Laird, & Cavallaro, 1999). It is also now clear that emotion-related bodily movements can also have cognitive effects consistent with the persons' emotional state. In the previously mentioned study by Keltner et al. (1993) people who adopted the facial expression and bodily posture characteristic of anger typically made the external-agency appraisals predicted by

¹⁸Laird and his associates (e.g., 1984; Duclos, Laird et al., 1989; Duclos & Laird, 2001) have repeatedly discussed the many reasons why it is implausible to say that the bodily feedback results obtained in their experiments were due to "demand" influences. Strack, Martin, and Stepper (1988) made much the same point about their research.

most appraisal accounts of anger, whereas the sad pose resulted in more situational attributions. Even memory can be influenced by the peripheral facial-muscular movements. In one experiment reported by Laird, Wagener, and colleagues (1982), the adoption of an angry facial expression while attempting to remember the sentences heard earlier led to the best recall when the material heard had been angry in nature. The affective match between the sentence content and the expression had facilitated the ease with which the sentence could be brought to mind.

Even less attention is given to the studies dealing with a bodily action-induced "mood contagion." There is now increasing evidence that one individual's emotion-related bodily movements can trigger that emotion in other persons (see Bargh & Chartrand, 1999, and Neumann & Strack, 2000, for a summary of relevant research).¹⁹ The intriguing experiments reported by Neumann and Strack (2000) demonstrated, for example, that a spoken statement whose content was affectively neutral but which was expressed in either a slightly happy or slightly sad tone of voice evoked a congruent affective state in the listeners. This "contagion" occurred, moreover, even though the listeners had not consciously wanted to share the speaker's emotion, had not devoted much of their cognitive resources to what was said so that they had not fully understood the content, and were unaware that their mood had been affected by the statement's emotional tone.

Laird and colleagues (e.g., Duclos, Laird et al., 1989, Duclos & Laird, 2001; Laird & Bresler, 1992) have consistently favored a self-perception interpretation of these bodily action effects, very much in keeping with Bem's (1972) self-perception perspective. Individuals taking up an emotion-like facial expression and/or body posture presumably detect the muscular changes that result, and then automatically and nonconsciously use these cues, together with cues from the surrounding situation, in forming their emotional experience. Laird (e.g., 1984; Duclos & Laird, 2001; Duclos, Laird et al., 1989; Laird & Bresler, 1992) has also proposed that there are individual differences in the degree to which the emotion-related muscular changes give rise to emotional experiences. Some people are more prone to rely on their inner sensations, i.e., their self-produced cues, in developing knowledge of what emotion they are feeling, whereas others rely more heavily on cues from the external circumstances.²⁰ These individual differences are stable over time and relate to other personal characteristics. In many of their experiments these researchers used a preliminary assessment to divide participants into a self-produced cue group or a situational cue group, and showed that the former people's reported emotions typically were more strongly affected by the muscular movements they made. As just one example, in the Flack et al. (1999) study, the persons whose emotions were most strongly influenced by their facial expressions were also the ones most affected by their postures. Duclos and Laird (2001) have argued that these differences in responsiveness to bodily cues can account for the inconsistent findings in studies as to whether expression inhibition can lessen emotional feelings

Even with all of these results consistent with Laird's self-perception thesis, I favor a more associationistic conception (that I believe is compatible with the self-perception idea), largely because of the voluminous literature regarding the role of associative processes in emotion arousal. In accord with Bower's (1981; also see Berkowitz, 2000) associative network view of mood-memory effects, I suggest that the various physiological, skeletal-muscular, experiential, and cognitive

¹⁹In terminology perhaps more familiar to cognitive psychologists, the line of reasoning favored by Neumann and Strack (2000) would say that the first individual's emotional expression activated a compatible action code in the observer which led to the arousal of the congruent feelings. The Bargh and Chartrand (1999) formulation is very similar and would hold that because of an "ideomotor" linkage the perception of the actor's expression automatically created a related behavioral tendency which produced the related feelings.

²⁰In the experiment by Duclos and Laird (2001) an imagery technique in which the participants had to imagine themselves in a particular emotional situation was more effective in arousing emotional feelings in the situational cue responders, whereas the emotional expression procedure did this more effectively in the self-produced cue responders.

components of an emotional syndrome are interconnected associatively so that the activation of any one component will spread to other components in proportion to the strength of the associations between them. Just as the arousal of a distinctive emotional feeling will tend to activate the cognitions with which it is linked (e.g., other-agency appraisals in the case of anger), a facial expression and/or bodily posture that is characteristic of a particular emotional state will activate the other components in that emotional syndrome. The consequence is that the performance of the emotion-related muscular movement will have widespread congruent physiological, experiential, and cognitive effects. Accordingly, I propose that those participants who were designated as self-produced cue users in the Laird studies were highly responsive to their facial/bodily sensations because they had fairly strong associative links among the various components of emotional syndromes. These associative connections were presumably weaker in those persons who were the situational cue users.

16.6 Conclusions

My critique of the causal propositions offered by several of the best known appraisal analyses is based largely on experimental findings. Importantly, many of the experimental results I summarized indicate that anger can arise even when a number of these appraisal features are not present. Further research, especially of a controlled nature, clearly is required to determine the conditions under which the appraisal propositions hold and what psychological processes operate on these occasions to produce the outcomes.

Experiments can also address some of the more specific uncertainties that were identified in the above review. I noted, for example, that there is some disagreement among appraisal proponents as to whether an anger-provoking occurrence must be viewed as illegitimate in some way, a violation of accepted standards of conduct (see Roseman et al., 1996). Several experiments have shown that events regarded as unjust can produce angry reactions (e.g., Baron et al., 1999; Weiss et al., 1999), but other controlled investigations indicate that even socially proper impediments to goal attainment can occasionally instigate anger/hostility (Berkowitz, 1989; Dill & Anderson, 1995). Sufficiently sophisticated experimental designs might well show why this inconsistency exists, what are the underlying factors that help determine when and why a perceived improper event will arouse anger.

The analysis of anger arousal presented here is a “bottom-up” formulation, and I suggest that appraisal conceptions of emotion would do well to devote more theoretical and research attention to “bottom-up” processes in general. Bodily feedback effects, I believe, provide a very good Example of these processes in operation. Whatever the terminology and/or concepts that are employed, however, the really important aspect has to do with the extended range of phenomena to be incorporated into theoretical analyses of emotion. This chapter encourages appraisal researchers to be more expansive to broaden their horizons, to widen the span of methods they employ in their investigations, and to increase the range of phenomena they study and incorporate into their analyses of emotions. Surely, a truly comprehensive psychological formulation of emotional experience and behavior must deal with more than self-reported responses to the kinds of remembered or imagined emotional episodes typically used in traditional appraisal research.

References

- Adelmann, P. K., & Zajonc, R. B. (1989). Facial efference and the experience of emotion. *Annual Review of Psychology*, 40, 249–280.
- Anderson, C. A., & Anderson, K. B. (1998). Temperature and aggression: Paradox, controversy, and a (fairly) clear picture. In R. G. Geen, & E. Donnerstein (Eds.), *Human aggression: Theories, research, and implications for social policy* (pp. 247–298). San Diego, CA: Academic Press.

- Averill, J. (1982). *Anger and aggression: An essay on emotion*. New York: Springer-Vela.
- Bargh, J. A., & Chartrand, T. L. (1999). The unbearable automaticity of being. *American Psychologist*, 54, 462–479.
- Baron, R. A., Neumann, J. H., & Geddes, D. (1999). Social and personal determinants of workplace aggression: Evidence for the impact of perceived injustice and Type A behavior pattern. *Aggressive Behavior*, 25, 281–296.
- Bem, D. J. (1972). Self-perception theory. In L. Berkowitz (Ed.), *Advances in experimental social psychology* (Vol. 6, pp. 2–62). New York: Academic Press.
- Berkowitz, L. (1983). Aversively stimulated aggression: Some parallels and differences in research with animals and humans. *American Psychologist*, 38, 1135–1144.
- Berkowitz, L. (1989). Frustration-aggression hypothesis: Examination and reformulation. *Psychological Bulletin*, 106, 59–73.
- Berkowitz, L. (1990). On the formation and regulation of anger and aggression: A cognitive-neoassociationistic analysis. *American Psychologist*, 45, 494–503.
- Berkowitz, L. (1993). *Aggression: Its causes, consequences, and control*. New York: McGraw-Hill.
- Berkowitz, L. (1998). Affective aggression: The role of stress, pain, and negative affect. In R. G. Geen, & E. Donnerstein (Eds.), *Human aggression: Theories, research, and implications for social policy* (pp. 49–72). San Diego, CA: Academic Press.
- Berkowitz, L. (1999). Anger. In T. Dalgleish, & M. Power (Eds.), *Handbook of cognition and emotion* (pp. 411–428). Chichester, UK/New York: Wiley.
- Berkowitz, L. (2000). *Causes and consequences of feelings*. Cambridge, UK: Cambridge University Press.
- Berkowitz, L. (2002). Affect, aggression, and antisocial behavior. In R. J. Davidson, K. R. Scherer, & H. H. Goldsmith (Eds.), *Handbook of affective sciences* (pp. 804–823). New York: Oxford University Press.
- Berkowitz, L., Cochran, S., & Embree, M. (1981). Physical pain and the goal of aversively stimulated aggression. *Journal of Personality and Social Psychology*, 40, 687–700.
- Berkowitz, L., & Harmon-Jones, E. (2004). Toward an understanding of the determinants of anger. *Emotion*, 4, 107–130.
- Bower, G. H. (1981). Mood and memory. *American Psychologist*, 36, 129–148.
- Catalano, R., Dooley, D., Novaco, R., Wilson, G., & Hough, R. (1993). Using ECA survey data to examine the effects of job layoffs on violent behavior. *Hospital and Community Psychology*, 44, 874–878.
- Clore, G. L., Ortony, A., Dienes, B., & Fujita, F. (1993). Where does anger dwell? In R. S. Wyer, Jr., & T. K. Srull (Eds.), *Advances in social cognition: Perspectives on anger and emotion* (Vol. 6, pp. 57–87). Hillsdale, NJ: Erlbaum.
- Dill, J. C., & Anderson, C. A. (1995). Effects of frustration justification on hostile aggression. *Aggressive Behavior*, 21, 359–369.
- Dollard, J., Doob, L., Miller, N., Mowrer, O., & Sears, R. (1939). *Frustration and aggression*. New Haven: Yale University Press.
- Donnerstein, E., & Wilson, D. W. (1976). Effects of noise and perceived control on ongoing and subsequent aggressive behavior. *Journal of Personality and Social Psychology*, 34, 774–781.
- Duclos, S. E., & Laird, J. D. (2001). The deliberate control of emotional experience through control of expressions. *Cognition and Emotion*, 15, 27–56.
- Duclos, S. E., Laird, J. D., Schneider, E., Sexter, M., Stern, L., & Van Lighten, O. (1989). Emotion-specific effects of facial expressions and postures on emotional experience. *Journal of Personality and Social Psychology*, 57, 100–108.
- Ekman, P. (1984). Expression and the nature of emotion. In K. Scherer, & P. Ekman (Eds.), *Approaches to emotion* (pp. 319–344). Hillsdale, NJ: Erlbaum.
- Ekman, P. (1993). Facial expression and emotion. *American Psychologist*, 48, 384–392.
- Ellsworth, P. C., & Scherer, K. R. (2003). Appraisal processes in emotion. In R. J. Davidson, H. Goldsmith, & K. R. Scherer (Eds.), *Handbook of the affective sciences*. UK: Oxford University Press: New York/Oxford.
- Ellsworth, P. C., & Smith, C. A. (1988). From appraisal to emotion: Differences among unpleasant feelings. *Motivation and Emotion*, 12, 271–302.
- Fernandez, E., & Turk, D. C. (1995). The scope and significance of anger in the experience of chronic pain. *Pain*, 61, 165–175.
- Flack, W. F., Jr., Laird, J. D., & Cavallaro, L. A. (1999). Separate and combined effects of facial expressions and bodily postures on emotional feelings. *European Journal of Social Psychology*, 29, 203–217.
- Frijda, N. H. (1993). The place of appraisal in emotion. *Cognition and Emotion*, 7, 357–387.
- Frijda, N. H., Kuipers, P., & ter Schure, E. (1989). Relations among emotion, appraisal, and emotional action readiness. *Journal of Personality and Social Psychology*, 57, 212–228.
- Frijda, N. H., & Zeelenberg, M. (2001). Appraisal: What is the dependent? In K. R. Scherer, A. Schorr, & T. Johnstone (Eds.), *Appraisal processes in emotion*. Oxford, UK/New York: Oxford University Press.

- Geen, R. G. (1968). Effects of frustration, attack, and prior training in aggressiveness upon aggressive behavior. *Journal of Personality and Social Psychology*, 9, 316–321.
- Geen, R. G. (1978). Effects of attack and uncontrollable noise on aggression. *Journal of Research in Personality*, 9, 270–281.
- Geen, R. G. (1998). Aggression and antisocial behavior. In D. T. Gilbert, S. T. Fiske, & G. Lindzey (Eds.), *The handbook of social psychology*. 4th ed (Vol. II, pp. 317–356). Boston, MA: McGraw-Hill.
- Gigerenzer, G. (1991). From tools to theories: A heuristic of discovery in cognitive psychology. *Psychological Review*, 98, 254–267.
- Green, D. P., Glaser, J., & Rich, A. (1998). From lynching to gay bashing: The elusive connection between economic conditions and hate crime. *Journal of Personality and Social Psychology*, 75, 82–92.
- Harmon-Jones, E., & Mills, J. (1999). *Cognitive dissonance: Progress on a pivotal theory in social psychology*. Washington, D.C.: American Psychological Association.
- Harmon-Jones, E., Sigelman, J. D., Bohlig, A., & Harmon-Jones, C. (2003). Anger, coping, and frontal cortical activity: The effect of coping potential on anger-induced left frontal activity. *Cognition and Emotion*, 17, 1–24.
- Hatch, J. P., Moore, P. J., Borcherding, S., Cyr-Provost, M., Boutros, N. N., & Seleshi, E. (1992). Electromyographic and affective responses of episodic tension-type headache patients and headache-free controls during stressful task performance. *Journal of Behavioral Medicine*, 15, 89–112.
- Izard, C. E. (1971). *The face of emotion*. New York: Appleton-Century-Crofts.
- Jo, E. (1993). *Combining physical sensations and ideas in the construction of emotional experiences*. Madison, WI: Unpublished doctoral dissertation, University of Wisconsin.
- Keltner, D., Ellsworth, P. C., & Edwards, K. (1993). Beyond simple pessimism: Effects of sadness and anger on social perception. *Journal of Personality and Social Psychology*, 64, 740–752.
- Laird, J. D., Wagener, J. J., Halal, M., & Szegda, M. (1982). Remembering what you felt: The effect of emotion on memory. *Journal of Personality and Social Psychology*, 42, 646–658.
- Laird, J. D. (1984). The real role of facial response in the experience of emotion: A reply to Tourangeau and Ellsworth, among others. *Journal of Personality and Social Psychology*, 47, 909–917.
- Laird, J. D., & Bresler, C. (1992). The process of emotional experience: A self-perception theory. In M. S. Clark (Ed.), *Review of personality and social psychology* (vol. 13, pp. 213–234). Newbury Park, CA: Sage.
- Lazarus, R. S. (1991). *Emotion and adaptation*. Oxford, UK/New York: Oxford University Press.
- Lerner, J. S., & Keltner, D. (2000). Beyond valence: Toward a model of emotion-specific influences on judgment and choice. *Cognition and Emotion*, 14, 473–493.
- Lerner, J. S., & Keltner, D. (2001). Fear, anger, and risk. *Journal of Personality and Social Psychology*, 81, 146–159.
- Leventhal, H., & Scherer, K. R. (1987). The relationship of emotion to cognition: A functional approach to a semantic controversy. *Cognition and Emotion*, 1, 3–28.
- Lewis, M. (1993). The development of anger and rage. In R. A. Glick, & S. P. Roose (Eds.), *Rage, power, and aggression* (pp. 148–168). New Haven: Yale University Press.
- Lindsay, J. J., & Anderson, C. A. (2000). From antecedent conditions to violent actions: A general affective aggression model. *Personality and Social Psychology Bulletin*, 26, 533–547.
- Mikula, G., Scherer, K. R., & Athenstaedt, U. (1998). The role of injustice in the elicitation of differential emotional reactions. *Personality and Social Psychology Bulletin*, 24, 769–783.
- Mikulincer, M. (1988). Reactance and helplessness following exposure to unsolvable problems: The effects of attributional style. *Journal of Personality and Social Psychology*, 54, 679–686.
- Miller, N. E. (1948). Theory and experiment relating psychoanalytic displacement to stimulus-response generalization. *Journal of Abnormal and Social Psychology*, 43, 155–178.
- Miller, I. W., & Norman, W. H. (1979). Learned helplessness in humans: A review and attribution theory model. *Psychological Bulletin*, 86, 93–118.
- Neumann, R. (2000). The causal influences of attributions on emotions: A procedural priming approach. *Psychological Science*, 11, 179–182.
- Neumann, R., & Strack, F. (2000). “Mood contagion”: The automatic transfer of mood between persons. *Journal of Personality and Social Psychology*, 79, 211–223.
- Oatley, K., & Jenkins, J. M. (1996). *Understanding emotions*. Cambridge, MA: Blackwell.
- Ortony, A., Clore, G. L., & Collins, A. (1988). *The cognitive structure of emotions*. New York: Cambridge U. Press.
- Parkinson, B. (1999). Relations and dissociations between appraisal and emotion ratings of reasonable and unreasonable anger and guilt. *Cognition and Emotion*, 13, 347–385.
- Parkinson, B., & Manstead, A. S. R. (1992). Appraisal as a cause of emotion. In M. S. Clark (Ed.), *Review of personality and social psychology* (Vol. 13, pp. 122–149). Newbury Park, CA: Sage. stories and social life. *Cognition and Emotion*, 7, 295–323.

- Quigley, B. M., & Tedeschi, J. T. (1996). Mediating effects of blame attributions on feelings of anger. *Personality and Social Psychology Bulletin*, 22, 1280–1288.
- Robinson, M. D., & Clore, G. L. (2001). Simulation, scenarios, and emotional appraisal: Testing the convergence of real and imagined reactions to emotional stimuli. *Personality and Social Psychology Bulletin*, 27, 1520–1532.
- Roseman, I. J. (1991). Appraisal determinants of discrete emotions. *Cognition and Emotion*, 5, 161–200.
- Roseman, I. J., Antoniou, A. A., & Jose, P. E. (1996). Appraisal determinants of emotions: Constructing a more accurate and comprehensive theory. *Cognition and Emotion*, 10, 241–277.
- Roseman, I. J., & Smith, C. A. (2001). Appraisal theory: Overview, assumptions, varieties, controversies. In K. R. Scherer, A. Schorr, & T. Johnstone (Eds.), *Appraisal processes in emotion*. UK/New York: Oxford University Press: Oxford.
- Roseman, I. J., Spindel, M. S., & Jose, P. E. (1990). Appraisals of emotion-eliciting events: Testing a theory of discrete emotions. *Journal of Personality and Social Psychology*, 59, 899–915.
- Roseman, I. J., Wiest, C., & Swartz, T. S. (1994). Phenomenology, behaviors and goals differentiate discrete emotions. *Journal of Personality and Social Psychology*, 67, 206–221.
- Russell, J. A., & Fehr, B. (1994). Fuzzy concepts in a fuzzy hierarchy: Varieties of anger. *Journal of Personality and Social Psychology*, 67, 186–205.
- Scherer, K. R. (1984). Emotion as a multicomponent process: A model and some cross-cultural data. In P. Shaver (Ed.), *Review of personality and social psychology*. Vol. 5 (pp. 37–63). CA: Sage: Newbury Park.
- Scherer, K. R. (1993). Studying the emotion-antecedent appraisal process: An expert system approach. *Cognition and Emotion*, 7, 325–355.
- Scherer, K. R. (1999). Appraisal theory. In T. Dalgleish, & M. Power (Eds.), *Handbook of cognition and emotion* (pp. 637–663). Chichester, UK/New York: Wiley.
- Scherer, K. R. (2001a). Appraisal considered as a process of multilevel sequential checking. In K. R. Scherer, A. Schorr, & T. Johnstone (Eds.), *Appraisal processes in emotion* (pp. 92–120). Oxford, UK/New York: Oxford University Press.
- Scherer, K. R. (2001b). The nature and study of appraisal: A review of the issues. In K. R. Scherer, A. Schorr, & T. Johnstone (Eds.), *Appraisal processes in emotion* (pp. 369–391). Oxford, UK/New York: Oxford University Press.
- Scherer, K. R., Schorr, A., & Johnstone, T. (2001). *Appraisal processes in emotion*. Oxford, UK/New York: Oxford University Press.
- Shaver, P., Schwartz, J., Kirson, D., & O'Connor, C. (1987). Emotion knowledge: Further exploration of a prototype approach. *Journal of Personality and Social Psychology*, 52, 1061–1086.
- Siemer, M. (2001). Mood-specific effects on appraisal and emotion judgements. *Cognition and Emotion*, 15, 453–485.
- Smith, C. A., & Ellsworth, P. C. (1985). Patterns of cognitive appraisal in emotion. *Journal of Personality and Social Psychology*, 48, 813–838.
- Smith, C. A., Haynes, K. N., Lazarus, R. S., & Pope, L. K. (1993). In search of the “hot” cognitions: Attributions, appraisals, and their relation to emotion. *Journal of Personality and Social Psychology*, 65, 916–929.
- Smith, C. A., & Kirby, L. D. (2000). Consequences require antecedents: Toward a process model of emotion elicitation. In J. P. Forgas (Ed.), *Feeling and thinking: The role of affect in social cognition* (pp. 83–106). Cambridge, UK/New York: Cambridge University Press.
- Smith, C. A., & Kirby, L. D. (2001). Toward delivering on the promise of appraisal theory. In K. R. Scherer, A. Schorr, & T. Johnstone (Eds.), *Appraisal processes in emotion*. UK/New York: Oxford University Press: Oxford.
- Smith, C. A., & Lazarus, R. S. (1993). Appraisal components, core relational themes, and the emotions. *Cognition and Emotion*, 7, 233–269.
- Solomon, R. C. (1993). The philosophy of emotions. In M. Lewis, & J. M. Haviland (Eds.), *Handbook of emotions* (pp. 3–15). New York/London: Guilford.
- Stein, N. L., & Levine, L. J. (1989). The causal organization of emotional knowledge: A developmental study. *Cognition and Emotion*, 3, 343–378.
- Stein, N. L., & Levine, L. J. (1990). Making sense out of emotion: The representation and use of goal-structured knowledge. In N. L. Stein, B. Leventhal, & T. Trabasso (Eds.), *Psychological and biological approaches to emotion* (pp. 45–73). Hillsdale, NJ: Erlbaum.
- Stein, N. L., & Levine, L. J. (1999). The early emergence of emotional understanding and appraisal: implications for theories of development. In T. Dalgleish, & M. J. Power (Eds.), *Handbook of cognition and emotion* (pp. 383–408). Chichester, UK/New York: Wiley.
- Stenberg, C. R., Campos, J. J., & Emde, R. N. (1983). The facial expression of anger in seven-month-old infants. *Child Development*, 54, 178–184.

- Strack, F., Martin, L. L., & Stepper, S. (1988). Inhibiting and facilitating conditions of facial expressions: A nonobtrusive test of the facial feedback hypothesis. *Journal of Personality and Social Psychology*, 54, 768–777.
- Sugimoto, J. D., & Oltjenbruns, K. A. (2001). The environment of death and its influence on police officers in the United States. *Omega: Journal of Death and Dying*, 43, 145–155.
- Tomkins, S. S. (1962, 1963). *Affect, imagery, consciousness*. New York: Springer.
- Toren, P., Wolmer, L., Weizman, R., Magal-Vardi, O., & Laor, N. (2002). Retraumatization of Israeli civilians during a reactivation of the Gulf War threat. *Journal of Nervous and Mental Disease*, 190, 43–45.
- Tourangeau, R., & Ellsworth, P. C. (1979). The role of facial response in the experience of emotion. *Journal of Personality and Social Psychology*, 37, 1519–1531.
- Venable, V. L., Carlson, C. R., & Wilson, J. (2001). The role of anger and depression in recurrent headache. *Headache*, 41, 21–30.
- Walters, R., & Brown, M. (1963). Studies of reinforcement of aggression. III. Transfer of responses to an interpersonal situation. *Child Development*, 34, 563–571.
- Weber, C., Arck, P., Mazurek, B., & Klapp, B. F. (2002). Impact of relaxation training on psychometric and immunologic parameters in tinnitus sufferers. *Journal of Psychosomatic Research*, 52, 29–33.
- Weiner, B., Graham, S., & Chandler, C. (1982). Pity, anger, and guilt: An attributional analysis. *Personality and Social Psychology Bulletin*, 8, 226–232.
- Weiss, H. M., Suckow, K., & Cropanzano, R. (1999). Effects of justice conditions on discrete emotions. *Journal of Applied Psychology*, 84, 786–794.
- Zillmann, D., Baron, R. A., & Tamborini, R. (1981). Social costs of smoking: Effects of tobacco smoke on hostile behavior. *Journal of Applied Social Psychology*, 11, 548–561.

Chapter 17

Fuel in the Fire: How Anger Impacts Judgment and Decision-Making

Paul M. Litvak, Jennifer S. Lerner, Larissa Z. Tiedens, and Katherine Shonk

Abstract In keeping with the handbook format, this chapter identifies four types of methods in the behavioral decision-making literature for detecting the influence of anger on judgments and choices. The types of methods include inferring the presence of anger from behavior, measuring naturally occurring anger or individual differences in anger, manipulating anger, and both measuring and manipulating anger. We discuss the strengths and weaknesses of each method and present evidence showing that the effects of anger often differ from those of other negative emotions. The chapter also introduces an overarching appraisal-tendency framework for predicting such effects and connects the framework to broader theories and associated mechanisms. Finally, we examine whether anger should be considered a positive emotion and propose that anger is experienced as pleasant when one is looking forward and unpleasant when one is reflecting back on the anger's source.

In this chapter, we focus on the judgment and decision-making outcomes of anger: how anger influences our perceptions, beliefs, ideas, reasoning, and ultimately our choices. We will review and synthesize an emerging literature that has explored how anger, as distinct from other emotions traditionally viewed as “negative,” affects judgment and decision-making. These cognitive effects of anger deserve attention for several reasons. First, anger is a commonly experienced emotion, at least among U.S. residents. In a survey of prior studies on anger, Averill (1982) concluded that “most people report becoming mildly to moderately angry anywhere from several times a day to several times a week” (p. 1146, see Chapter 19).¹ Similarly, in a nationally representative sample, Lerner, Gonzalez, Small, and Fischhoff (2003) identified anger as the most commonly experienced emotion experienced by U.S. citizens in response to the 9/11 terrorist attacks, and Fischhoff, Gonzalez, Lerner, and Small (2005) found that the same pattern held a year later with the same sample.

Second, displays of anger seize our attention (Solomon, 1990; Tavris, 1989). Hansen and Hansen (1988), for example, have demonstrated the “anger superiority effect,” or the tendency for people to identify angry faces more quickly and accurately than other emotion expressions. Angry expressers

J.S. Lerner (✉)
Harvard University, Cambridge, MA 02138, USA
e-mail: jennifer_lerner@harvard.edu

This chapter was adapted from Lerner and Tiedens, L. Z. (2006). Portrait of the angry decision maker: How appraisal tendencies shape anger's influence on cognition. *Journal of Behavioral Decision Making* (Special Issue on Emotion and Decision Making), 19, 115–137.

¹Regional variations may amplify or attenuate the frequency. Individuals in the southern United States, for example, tend to uphold a “culture of honor” (Cohen, Nisbett, Bowdle, & Schwarz, 1996), which includes lower thresholds for registering “a demeaning offense against me or mine” – a key trigger for anger (Lazarus, 1991a, p. 122).

are implicitly perceived as threatening, competent, powerful, and dominant, while sad expressers, by comparison, are perceived as likable, submissive, and in need of help (Clark, Pataki, & Carver, 1996; Tiedens, 2001a). Even 10-week-old infants respond differently to angry faces than to sad faces (Haviland & Lelwica, 1987). Thus, anger is likely to be a frequently used judgment cue, especially at the implicit level.

Third, once activated, anger can color people's perceptions, form their decisions, and guide their behavior while they remain angry, regardless of whether the decisions at hand are related to the source of their anger. In the aftermath of the September 11th attacks, experimentally inducing anger not only influenced U.S. citizens' risk perceptions about terror-related events (e.g., being attacked) but also their perceptions about routine events (such as getting the flu) and their policy preferences concerning matters of life and death (Lerner et al., 2003). Anger makes people indiscriminately punitive (Goldberg, Lerner, & Tetlock, 1999; Lerner, Goldberg, & Tetlock, 1998), indiscriminately optimistic about their own chances of success (Fischhoff et al., 2005; Lerner et al., 2003; Lerner & Keltner 2000, 2001), careless in their thought (Bodenhausen, Sheppard, & Kramer, 1994; Lerner et al., 1998; Small & Lerner, 2005; Tiedens, 2001b; Tiedens & Linton, 2001), and eager to take action (Harmon-Jones, Sigelman, Bohlig, & Harmon-Jones, 2003; Mackie, Devos, & Smith, 2000), effects we will review below.

Given that anger has the potential to grip a nation over a sustained period (Fischhoff et al., 2005; Lerner et al., 2003), it is important to understand how it may shape individual choices over time. By shaping basic cognitive and social processes, anger shapes the decisions we make and the lives we lead. In this chapter, we will review the impact of anger on judgment and decision-making. We begin by examining how anger has been studied by judgment and decision-making researchers and present the Appraisal-Tendency Framework as a means of predicting and organizing the effects of anger on cognition. In addition, we will review the evidence concerning the uniqueness of anger's effects on judgment and decision-making and explore possible mechanisms underlying these effects. Finally, citing evidence to the contrary, we conclude by presenting the question of whether anger is truly a negative emotion.

17.1 Anger and the Appraisal-Tendency Framework

More than two decades of research have supported the intuition that being in a globally negative mood can lead a person to form relatively pessimistic expectations, whereas being in a globally positive mood can lead one to form relatively optimistic expectations (for a review, see Forgas, 2003). For example, one influential study found that participants induced to feel negative affect consistently made more pessimistic estimates about frequencies of death than did participants induced to feel positive affect (Johnson & Tversky, 1983). This prototypic valence finding – that the presence of a (negative or positive) mood or disposition increases frequency estimates for similarly valenced events – helped to launch the field of affect and judgment and to demonstrate the replicability of effects across diverse tasks (Bower, 1991; Isen, Shalker, Clark, & Karp, 1978; Mayer & Hanson, 1995; Schwarz & Clore, 1983; Wright & Bower, 1992). Indeed, the emerging literature led Forgas to conclude in his authoritative chapter for the *Handbook of Affective Science* that "...most of the research suggests a fundamental affect-congruent pattern: positive affect improves, and negative affect impairs, the value of self-conceptions" (2003, p. 602).

Yet recent studies question the assumption that all negative emotions fit such valence-congruent patterns. Specifically, studies examining emotion effects on attribution, evaluation, and judgments involving risk all reveal that anger has distinct effects. In fact, anger can actually enhance self-conceptions despite being widely viewed as a negative emotion (Lerner & Keltner, 2000).

To distinguish the effects of specific emotions on judgment and decision-making Lerner and Keltner (2000, 2001) proposed the appraisal-tendency framework (ATF). The ATF makes two broad theoretical assumptions. First, it assumes that a discrete set of cognitive dimensions differentiates emotional experience and effects (Lazarus, 1994; Ortony, Clore, & Collins, 1988; Roseman, 1984; Scherer, 2001; C. A. Smith & Ellsworth, 1985; Weiner, 1980). For example, in an empirical examination of appraisal dimensions of emotions, Smith and Ellsworth (1985) identified six cognitive dimensions that define the patterns of appraisal underlying different emotions: certainty, pleasantness, attentional activity, control, anticipated effort, and responsibility. Each emotion was found to be defined by central dimensions that characterize its core meaning or theme, for example, anger being defined by a sense of certainty and individual control along with other-responsibility (Lazarus, 1991b; C. A. Smith & Ellsworth, 1985). For example, one becomes angry when appraising that a person as opposed to natural forces (individual control) clearly (sense of certainty) and with sound mind (other-responsibility) stole a purse. By contrast, one becomes sad if natural forces rained on the purse and ruined it.

Second, the ATF assumes that emotions automatically trigger a set of responses (physiology, behavior, experience, and communication) that enable an individual to deal quickly with problems or opportunities (Frijda, 1988; Keltner & Gross, 1999; Levenson, 1994; Oatley & Jenkins, 1992; Plutchik, 1979).² Even without accompanying thought, emotions trigger action toward implicit goals – what Frijda (1986) has been called “action tendencies.” These tendencies depend not only on an emotion’s intensity but also on its qualitative character.

The ATF predicts that each emotion carries with it motivational properties that fuel carryover to subsequent judgments and decisions. Emotions not only can arise from but also give rise to an implicit cognitive predisposition to appraise future events in line with an “appraisal tendency,” or a goal-directed process through which an emotion affects judgment and choice until the emotion-eliciting problem is resolved. Although such appraisals are tailored to help a person respond to the event that evoked the emotion, they persist beyond the eliciting situation, becoming an unconscious perceptual lens for interpreting subsequent judgments and choices.

Rather than shutting down thought, emotions direct attention, memory, and judgment toward the emotion-eliciting event (Johnson-Laird & Oatley, 1992; Lazarus, 1991b; Schwarz, 1990; Simon, 1967; Tooby & Cosmides, 1990) and even to unrelated events – what is often called the carryover of *incidental emotion* (Bodenhausen, 1993; Loewenstein & Lerner, 2003). For example, incidental anger triggered in one situation can automatically elicit a motive to blame in other situations (Quigley & Tedeschi, 1996). Interestingly, the effects of incidental emotion can be so strong that they drive behavior even when people have a financial incentive to disregard irrelevant influences on their judgment (Lerner, Small, & Loewenstein, 2004).

Because appraisals are a cognitive component of emotion and because most judgments and decisions involve cognitive processes, the ATF is useful for the study of the effects of specific emotions on judgment and choice. Appraisals are especially likely to play a major role in novel, complex situations in which individuals must weigh a number of factors, such as situational constraints. Appraisals differentiate emotions more precisely than valence approaches and also break emotions into cognitive dimensions that may help to map emotions onto judgment and decision-making processes.

²We thank a reviewer from Lerner and Tiedens (2006) for suggesting that the ATF rests squarely within diverse streams of research showing emotion consonance. For example, feeling an emotion can evoke consonant facial and other bodily expressions (“Method Acting”). Behavioral expressions of emotions can evoke the associated feelings and appraisals (Cacioppo, Priester, & Berntson, 1993; Musch & Klauer, 2004). In addition, the sociological literature on emotion management (Hochschild, 1983) reveals the stress associated with attempting to block emotion-consonant behavior.

Identifying these dimensions is crucial to understanding the nature of emotional experience and to understanding the effects of specific emotions on judgment and decision-making.

The ATF points to a clear empirical strategy: research should compare emotions that are highly differentiated in their appraisal themes on judgments/choices that relate to that appraisal theme. For example, because the cognitive appraisal dimension of responsibility shares a conceptual theme with blame judgments, researchers interested in studying emotion effects on blame could contrast emotions on opposite poles of the responsibility dimension, such as sadness (situational responsibility) and anger (individual responsibility) (Ellsworth & Smith, 1988; C. A. Smith & Ellsworth, 1985). Similarly, based on the ATF, one could predict that fear and anger trigger differential cortisol responses to a stressor (Lerner, Dahl, Hariri, & Taylor, 2007) because the two emotions trigger different appraisals of certainty and individual control (Lerner & Keltner, 2001). According to the ATF, biological stress responses may depend more on whether an emotion is associated with a sense of individual control and predictability rather than whether an emotion is associated with negativity. These examples suggest that it is possible to use the ATF to make systematic predictions about the precise ways in which anger will differ from other emotions of the same valence.

Indeed, a remarkably consistent picture of anger has emerged from studies that have investigated the experience of anger and its related appraisals (e.g., Lazarus, 1991a; Ortony et al., 1988; Roseman, 1984, 1991; Scherer, 1999, 2001; Weiner, 1980, 1986). Specifically, anger has been associated with a sense that the self (or someone the self cares about) has been offended or injured (Lazarus, 1991a), with a sense of certainty or confidence about the angering event and what caused it, and with the belief that another person (as opposed to the situation or the self) was responsible for the event and with the notion that one can still influence the situation or cope with it (e.g., Lazarus, 1991a; Ortony et al., 1988; Roseman, 1984, 1991; Scherer, 1999, 2001; Weiner, 1980, 1986). By contrast, people can have completely different sets of appraisals about negative events more generally and thus experience different negative emotions. For example, when someone blames a negative event on situational forces, she is more likely to feel sad than angry. If someone feels responsible for a negative event, he may feel guilt and shame rather than anger (Neumann, 2000). And when someone feels uncertain or lacks confidence about the cause of a negative event, she is likely to experience fear and anxiety rather than anger.

Notably, emotions, including anger, may arise in any number of ways, including relatively noncognitive routes, such as bodily feedback or unconscious priming (Berkowitz & Harmon-Jones, 2004; Keltner, Ellsworth, & Edwards, 1993; Parkinson, 1996). Similarly, in the case of well-practiced anger, as in frequently repeated familial situations, anger might become automatic and require little appraisal. However, even when anger or another emotion is not elicited through an appraisal process it can still activate the appraisal system, resulting in appraisal-consistent judgment. For example, Keltner, Locke, and Audrain (1993) showed that emotions induced via facial muscle movements gave rise to appraisal tendencies that shaped subsequent judgments (cf., Berkowitz, Chapter 16). More generally, emotions and appraisals have a positive feedback relationship, each making the other more likely. The more anger one feels, for example, the more one perceives others to be responsible for a negative event; the more one perceives others as responsible for a negative event, the more anger one feels (Quigley & Tedeschi, 1996).³

³Because of the recursive relationship of appraisals and emotion, we believe that in most cases, fully experiencing an emotion means also experiencing the cognitive appraisals that comprise that emotional state (Clore, 1994; Frijda, 1994; Lazarus, 1994). It is important to point out, however, that a primary causal role for appraisals in emotion is not a necessary condition for the ATF. It is sufficient to assume that a discrete set of cognitive dimensions differentiates emotional experience and effects (as is widely documented: see review by Ellsworth & Scherer, 2003).

Appraisals involve themes that have been central to decision-making research, including our perceptions of the likelihood of various events and how we assign responsibility, blame, and causality. We argue that the appraisals associated with emotions will influence such judgments. Because the experience of anger (but not of some other negative emotions) involves a sense of certainty and control of or responsibility for a negative event, people's perceptions about these aspects of subsequent situations are colored by their experience of anger. And because anger has unique associations with certainty, control, and responsibility, its effects on judgments relevant to these dimensions will be distinct from other negative emotions.

Turning to the motivational properties of anger, in their investigation of action tendencies, Frijda, Kuipers, and ter Schure (1989) found that anger was associated with a desire to change a situation for the better, if sometimes through destructive means such as fighting. The readiness to fight manifests itself biologically as well; some of anger's response tendencies are associated with relative left frontal hemispheric activation in the brain, a pattern characteristic of approach motivation (Harmon-Jones, 2004, 2007; Harmon-Jones & Sigelman, 2001). This approach tendency is sometimes also associated with a range of other changes in peripheral physiology that might prepare one to fight, such as blood flow to the hands⁴ (Ekman, Levenson, & Friesen, 1983).

17.2 How Decision Researchers Have Studied Anger

Just as anger has a variety of effects on cognition more broadly, psychologists and behavioral economists have viewed anger in a variety of ways. Decision researchers have used four methodological strategies to study anger, ranging from simply inferring the presence of the emotion to realistically manipulating anger (see Table 17.1 for examples).

Table 17.1 Strategies for determining the locus of anger's effect on judgment and decision-making

Strategy	Study	Effect of anger
Infer anger	Rabin (1993)	The desire "to hurt those who hurt them" drives the rejection of unfair offers in the ultimatum game
Measure anger	Lerner and Keltner (2001) (Study 1 and 2)	Relative to measured dispositional fear, dispositional anger is associated with risky choices and optimistic perceptions of risk
Manipulate anger	Lerner et al. (1998)	Relative to neutral emotion, induced anger activated more punitive attributions (e.g., amount of blame, harsher punishment, and heuristic processing (i.e., a reduction in the number of diagnostic cues used) in fictional tort cases
Measure and manipulate anger	Lerner et al. (2003)	Relative to naturally occurring anxiety, naturally occurring anger predicted optimistic perceptions of risks related to terrorism within the year following 9/11. Relative to induced fear, induced anger activated optimistic perceptions of risks related to terrorism within the year following 9/11
	Lerner et al. (2007)	Exposing subjects to the Trier Social Stress Test revealed a unique physiological response for angry individuals

⁴The evidence for increased blood flow to the hands is a matter of some debate, however. More research is needed to fully resolve this issue.

17.2.1 Inferring the Presence of Anger

Early judgment and decision-making research looked at anger as a possible mechanism to help explain certain deviations from rational, self-interested behavior. Rather than measuring or manipulating anger, scientists made inferences about anger and used it as a construct in theory building. To take one example, researchers used anger to explain why individuals are willing to forgo economic gain when rejecting unfair offers in the “ultimatum game” (Guth, Schmittberger, & Schwarze, 1982). In this two-player game, player 1 proposes how to divide a sum of money, typically \$10, and player 2 decides whether to accept or reject player 1’s offer. If player 2 rejects player 1’s offer, neither player receives any money. Researchers found that player 2s in this game typically reject unfair offers of \$1 or \$2 for themselves and \$9 or \$8 for player 1s, respectively; (for more, see Camerer, 2001). According to rational choice theory, such decisions are irrational, since people should always prefer some amount of money over no money at all. Theorists have pointed to anger, and an attendant desire to harm the proposer, to explain this behavior. As Rabin (1993) puts it, “If somebody is being mean to you, fairness allows – and vindictiveness dictates – that you be mean to him. Clearly, these emotions have economic implications.”

17.2.2 Measuring Anger

A second approach to investigating the influence of anger on judgment and decision-making has been to measure some correlate of anger and then correlate that measure with a subsequent behavior. For example, a study explicitly had subjects engage in an open-ended self-report of their reactions and feelings immediately following an unfair offer in the ultimatum game (Pillutla & Murnighan, 1996). Having subjects self-report their emotions prior to the dependent variable of interest (here the choice to accept or reject the offer) is generally not preferable, because labeling a target emotion can reduce its impact on a subsequent judgment (Keltner et al., 1993; Schwarz & Clore, 1983). While the authors use an open-ended response in part to mitigate this effect, it is possible that the self-report did have an impact. Indeed, the rejection rate of unfair offers (between 5 and 10% of the total sum) in the condition that replicates the basic ultimatum game is close to 44%, while in most studies the rejection rate is much closer to 100% (Camerer, 2001). Thus, although measuring the emotion did demonstrate that feeling anger in response to unfair offers is linked to rejecting the offer, the act of measurement still had an effect.

In another study, Lerner and Keltner (2001) presented study participants with Kahneman and Tversky’s Asian disease problem (Tversky & Kahneman, 1981), which required them to choose between (1) a “sure thing,” or an option in which a certain number of lives would be saved or lost, depending on how the option was framed; and (2) a gamble, or an option in which there was a smaller chance of saving more lives and a larger chance of saving no lives at all. Subjects self-reported their dispositional tendency to become afraid and angry. Lerner and Keltner found that regardless of the framing of the task, dispositionally fearful participants tended to make risk-averse choices, while dispositionally angry participants tended to make risk-seeking choices. Although the researchers could only correlate the emotion with the judgment, they did find that the strength of that relationship could overwhelm the effect of the framing manipulation on judgment. Thus, merely measuring emotion can yield useful insight.

17.2.3 Manipulating Anger

A more powerful strategy for examining anger is to elicit and then directly manipulate the emotion. In many studies, researchers have elicited anger by asking participants to vividly imagine an angering

experience, such as being derogated unfairly by a teaching assistant (Keltner et al., 1993). More recently, researchers have asked participants to write about an anger-eliciting situation (Lerner & Keltner, 2001) or have combined this type of writing task with an accompanying video induction (Gross & Levenson, 1995), such as a clip of someone being abused by a bully. Notably, studies of the effects of anger on judgment do not simultaneously manipulate anger and elicit self-reported anger, as doing so could attenuate the effect of anger on the subsequent judgment (Keltner et al., 1993).

17.2.4 Manipulating and Measuring Anger

To date, the most sophisticated method of examining the effects of anger on judgment and decision-making involves both manipulating and measuring the emotion. In one experiment (Lerner et al., 2007), subjects engaged in a stress task that required them to rapidly count down from a large number by 13. Before and after the task, experimenters collected samples of subjects' cortisol, a physiological marker of stress. The cortisol levels were correlated with the coded facial expressions subjects made during the stress task. As compared to fearful expressions, angry facial expressions were related to a unique physiological stress response. Thus, the experiment demonstrates how anger can be both manipulated (in the stress task) and measured (in facial expressions) to predict a subsequent response (decreasing cortisol as anger expression increased).

Similarly, neuroeconomists who have exposed subjects to unfair offers in the ultimatum game are manipulating anger with the goal of examining the neurological basis of subjects' choices. These studies use functional magnetic resonance imaging to reveal participants' brain activity when they receive unfair offers and decide whether to accept or reject them (Knoch, Pascual-Leone, Meyer, Treyer, & Fehr, 2006; Sanfey, Rilling, Aronson, Nystrom, & Cohen, 2003). Importantly, these studies do not localize the subjective experience of anger, but rather identify the brain structures and cognitive process that underlie the decision-making that follows from the angering experience. Consequently, the use of these convergent techniques can integrate our understanding of the cognitive and physiological dimensions of anger.

17.3 The Unique Effects of Anger on Judgment and Decision-Making

Anger has two primary types of effects on judgment and decision-making: outcome effects and process effects (see Table 17.2).

Table 17.2 Influences of anger on judgment and decision-making

Response tendency	Study	Impact of emotion
Attributions of causality	Keltner et al. (1993) ^b (Study 1 and 2)	Relative to sad people, angry people regarded dispositional and responsibility attributions as more likely and dispositional forces as more responsible for an ambiguous social event
	Quigley & Tedeschi (1996) ^b	Feelings of anger and thoughts of blame regarding a situation where someone harmed the participant escalated in a recursive loop, such that the more anger one experienced, the more blame one placed on the perpetrator, and vice versa

Table 17.2 (continued)

Response tendency	Study	Impact of emotion
Evaluations and attitudes	Goldberg et al. (1999) ^b	Relative to neutral emotion, anger activated more punitive attributional heuristics for inferring responsibility of harm, but only when the original source of the person's anger went unpunished (i.e., people relied on their own anger from normatively unrelated events when punishing a defendant in fictional tort cases)
	Mackie et al. (2000) ^a (Study 1 and 2)	Relative to fear, when the ingroup was considered strong, anger toward out-group members increased, as did the desire to take action toward out-group members
	DeSteno et al. (2004) ^b	Relative to sadness and neutral emotion, angry participants were slower to associate positive traits than negative traits with members of an out-group
Perceptions of risk	Dunn & Schweitzer (2005) ^b	Relative to sadness, guilt, gratitude, and pride, angry participants were less likely to trust others
	Lerner & Keltner (2000) ^a	Relative to fear, anger was associated with optimistic perceptions of future risk regarding the number of yearly deaths in the United States across various events (e.g., brain cancer, strokes, floods)
	Lerner & Keltner (2001) ^a (Study 1 and 2)	Relative to fearful people, angry people were more likely to make risk-seeking choices. In contrast to fearful people, happy and angry people held optimistic beliefs about experiencing future life events (e.g., heart attack at 50, developing gum problems, marrying someone wealthy)
	Lerner & Keltner (2001) ^b (Study 4)	Relative to fear, anger activated optimistic beliefs about experiencing future life events (e.g., heart attack at 50, developing gum problems, marrying someone wealthy)
	Lerner et al. (2003) ^{a,b}	Relative to naturally occurring anxiety, naturally occurring anger predicted optimistic perceptions of risks related to terrorism within the year following 9/11. Relative to induced fear, induced anger activated optimistic perceptions of risks related to terrorism within the year following 9/11
	Hemenover & Zhang (2004) ^b	Relative to neutral emotion, anger activated a defensive optimism that de-emphasized the importance and impact of negative events (i.e., two hypothetical stressors that participants were asked to imagine had happened to them already)
Attention effects	Fischhoff et al. (2005) ^b	Relative to fear and neutral emotion, anger activated optimistic perceptions for memories of terrorism-related risk judgments made after 9/11, judgments of what those risks really had been over the year after 9/11, and within the subsequent year (2002)
	DeSteno et al. (2000) ^b (Study 1)	Relative to sadness, anger increased likelihood estimates of angering events (e.g., intentionally being sold a "lemon" by a used car dealer) but not saddening events (e.g., a best friend moving away)
	DeSteno et al. (2004) ^b	Relative to sadness, anger activated perceptions that angry arguments (e.g., increased traffic delays) regarding an appeal to increase the city sales tax were more persuasive than sad arguments (e.g., suffering of special-needs infants)
Depth of processing	Bodenhausen et al. (1994) ^b	Relative to sadness and neutral emotion, anger activated heuristic processing (e.g., more stereotypic judgments, less attention paid to the quality of the arguments, and more attention to the superficial cues of the message)

Table 17.2 (continued)

Response tendency	Study	Impact of emotion
	Lerner et al. (1998) ^b	Relative to neutral emotion, anger activated more punitive attributions (e.g., amount of blame), harsher punishment, and heuristic processing (i.e., a reduction in the number of diagnostic cues used) in fictional tort cases
	Tiedens (2001b) ^b	Relative to sadness, happiness and neutral emotion, anger activated heuristic processing (e.g., use of chronically accessible scripts) and hostile inferences for aggressive (but not nonaggressive) participants
	Tiedens & Linton (2001) ^b (Study 2)	Relative to worry, anger activated heuristic processing (e.g., greater reliance on the superficial cues of the message and less attention to the argument quality)
	Small & Lerner (2005) ^b	Relative to sadness and neutral emotion, anger activated decisions to provide less public welfare assistance to welfare recipients unless participants were under cognitive load, in which case no difference between sadness and anger emerged

Note: All studies in this table have either directly measured or manipulated anger.

^a Indicates emotion was measured.

^b Indicates emotion was manipulated.

17.3.1 Outcome Effects of Anger

The first category emphasizes the *outcome* of judgments and choices, examining such questions as whether specific negative emotions trigger higher-risk estimates than other negative emotions, and if so, why.

17.3.1.1 Effects on Attribution and Evaluation

Research has revealed pervasive carryover effects of anger on attributions of causality, blame, and evaluations, effects that often diverge from the effects of other negative emotions (e.g., sadness and fear) on these same outcomes. Diverging from the valence-based paradigm described above, Keltner et al. (1993) asked whether it was possible for negative emotions to elicit effects other than undifferentiated pessimism. The researchers manipulated sadness and anger by presenting emotionally charged vignettes or by shaping participants' faces (unbeknownst to them) into prototypic expressions of the target emotion. Later, when asked to make judgments and/or choices concerning causality, sad participants perceived situationally caused negative events as more likely than did angry participants. In addition, sad participants perceived situational forces as more responsible for ambiguous events than did angry participants, who tended to attribute blame to another individual.

The results were consistent with the idea that the original appraisal patterns associated with each emotion triggered distinct appraisal tendencies in the subsequent judgments. That is, sadness appeared to not only co-occur with appraisals of situational control in the immediate situation, but also to trigger continuing perceptions of situational control even in novel situations. Anger co-occurred with appraisals of individual control and triggered continuing perceptions of such control. These studies demonstrated for the first time that when negative emotions carry over to judgment, they do not necessarily trigger an undifferentiated negative outlook (or mood congruency). Rather, at least in the case of anger and sadness, they have unique (and in this case, opposing) effects.

Other studies have further demonstrated the tendency for incidental anger to trigger attributions of individual blame. For example, relative to participants in a neutral state, participants induced to feel anger made more punitive attributions to a defendant and prescribed more punishment in a series of fictional tort cases even though the original source of the anger had nothing to do with the defendants in the tort cases (Goldberg et al., 1999; Lerner et al., 1998). This blaming tendency can be pernicious. As noted, feelings of anger and thoughts of blame can escalate in a positive feedback loop (Berkowitz, 1990; Quigley & Tedeschi, 1996). The more anger, the more blame placed on others, and vice versa.

These tendencies may be especially deleterious in interpersonal and intergroup relations. Recent research showed that incidental anger (created through movies, readings, and memories of anger-inducing events) seeped over to employees' judgments of their coworkers and acquaintances. Compared to happy and sad participants, angry participants felt less trusting of these coworkers, though they played no role in evoking the employees' anger. Consistent with the Appraisal-Tendency Framework, participants ratings of individuals' control of their own actions mediated the participants' lack of trust (Dunn & Schweitzer, 2005). Additionally, the mere experience of anger can activate precursors to prejudice. DeSteno, Dasgupta, Bartlett, and Cajric (2004) have shown, for example, that people in an angry state are slower to associate positive attributes than negative attributes with members of a group to which they do not belong. Importantly, people in a sad state do not show this same out-group prejudice. Along the same lines, when individuals consider their in-group to be strong, they feel greater anger in the presence of an out-group and a greater desire to take action against that out-group (Mackie et al., 2000). By contrast, though fear is also experienced in the presence of an out-group, fear, unlike anger, does not elicit the desire to take action against or move away from the out-group.

Interestingly, while anger can erode personal relationships, as Tiedens (2001a) demonstrated, being perceived as angry can also enhance one's social status, which may in part explain the persistence of anger's expression in organizational settings. Consequently, while anger often leads to biased judgments of others, those judgments also can be reinforced by the situation, leaving open the possibility that biased social judgments may be rational in a broader sense.

17.3.1.2 Effects on Risk Perception and Risk Preference

Lerner and Keltner (2000, 2001) originally used the ATF to examine emotion-based differences in judgments and choices involving risk. Anger and fear, as outlined earlier, differ markedly in their appraisal themes of certainty and control. Certainty and control, in turn, determine judgments of two types of risk: "unknown risk" (defined by hazards judged to be uncertain) and "dread risk" (defined by the perceived lack of individual control over hazards and the catastrophic potential of hazards) (McDaniels, Axelrod, Cavanagh, & Slovic, 1997; Slovic, 1987). Fear and anger, the researchers reasoned, should therefore exert different influences upon risk perception and preference. Indeed, the results of their initial tests found that fearful people made pessimistic risk assessments, whereas angry people made optimistic risk assessments (Lerner & Keltner, 2000, 2001). This finding has persisted regardless of the methods used in risk studies, such as showing movies, asking participants to recall prior events, or measuring self-reports of naturally occurring emotional experience (Fischhoff et al., 2005; Lerner et al., 2003; Lerner & Keltner, 2000, 2001).

In managerial settings where judgments can either lead to overly risk-averse or overly risk-seeking decisions (Kahneman & Lovallo, 1993), angry decision-making might lead to better or worse outcomes relative to a neutral affective state. Because anger exacerbates risk seeking and causes people to perceive less risk, anger could produce better judgments and choices than neutrality in situations where risk aversion is inappropriate. For example, when playing poker against

a small number of opponents (e.g., two), or when deciding how to succeed at an entrepreneurial venture, excessive risk aversion can often lead to poorer outcomes. This bears out in the lab as well; when individuals in a neutral affective state engage in the Balloon Analogue Risk Task, an externally validated measure of risk taking, they tend to adopt a more risk-averse strategy than the strategy that maximizes expected value (Lejuez et al., 2002). In a pilot study, two of the authors of this chapter (Lerner and Litvak) compared the performance of angry and neutral individuals' performance on the BART. Angry subjects were more risk seeking, and thus closer to the strategy that would maximize value. Thus, it is vital to characterize the specific context of risk-taking decisions facing angry individuals.

Notably, anger produced in one situation can carry over to a wide range of new situations, increasing both optimistic expectations for one's future and the likelihood of making risk-seeking choices; on the other hand, when fear is carried over, it leads to more pessimistic expectations and more risk-avoidant choices (Lerner et al., 2003). Moreover, recent work reveals that these cognitive appraisals also influence perceptions of one's own lived past and the concrete outcomes it yielded (Fischhoff et al., 2005).

Path-analytic models reveal that, in effect, fear and anger create opposing perceptual lenses, or appraisal tendencies; anger increases perceived control and certainty, and fear decreases such perceptions (Lerner & Keltner, 2000). Biological correlates of the anger-optimism link are also beginning to be understood. Lerner and colleagues (Lerner et al., 2007) have found that facial expressions of anger in response to a stressful task correlated with decreasing stress-hormone secretion, suggesting that the feelings of control associated with anger are adaptive under certain stressful circumstances.⁵

The optimism elicited by anger occurs not only in a relative sense (when compared to other negative emotions) but also in an absolute sense. Recent research shows that angry and happy individuals produce similar levels of optimism about the self (Lerner & Keltner, 2001). Moreover, these effects appear when participants consider both the likelihood of future events and negative events from the past. In the latter case, anger elicits a kind of "defensive optimism," in which angry people systematically de-emphasize the importance and potential impact of the negative events on the self (Hemenover & Zhang, 2004). Finally, these effects appear even when angry subjects rate the likelihood of events for which anger is a predisposing factor. That is, even though chronically angry people are more likely to have cardiovascular problems (Fredrickson et al., 2000; Williams et al., 2000), experience divorce, and have difficulty at work (Caspi, Elder, & Bem, 1987), angry people rate themselves as significantly *less* likely than the average person to experience these problems (Lerner et al., 2003; Lerner & Keltner, 2000, 2001).

⁵On the surface, the results could seem to conflict with research relating dispositional anger to enhanced stress reactivity and to stress-related disorders, such as coronary heart disease (for review, see Siegman & Smith, 1994). Anger, however, is heterogeneous (Harmon-Jones et al., 2003). Whereas behavioral medicine studies have typically found cardiovascular correlates with the intensity of a chronic dispositional tendency to experience explosive and violent anger (for example, see Spielberger, 1996), the Lerner et al. study found cardiovascular and cortisol correlates with the duration of situation-specific facial expressions of anger. It is important to note these differences. It may be that certain kinds of anger are adaptive, while others are not. Specifically, a low-intensity, controlled anger expression may be adaptive in a stress-challenge task with a pesky experimenter. Feeling a sense of indignation in the face of annoying badgering can be seen as reasonable. It is probably not adaptive, however, to chronically approach the world with a hostile edge. In sum, new results on anger imply the need to expand investigations of anger and biological stress responses by looking at anger not merely as a chronic dispositional quality, but also as a situation-specific behavioral response that may be justified and even adaptive under certain circumstances.

17.3.2 *Process Effects of Anger*

Anger appears to have unique effects on what people pay attention to and how much cognitive effort they expend in processing stimuli.

17.3.2.1 *Attention Effects*

Researchers have found that people selectively attend to and recall stimuli that have content or themes similar to the emotion they were experiencing prior to stimuli exposure. Such selective attention effects are not limited to valence, but occur even for specific emotions (Niedenthal, Halberstadt, & Setterlund, 1997). For example, Niedenthal et al. (1997) showed that sadness increased processing of sad words but not of angry words, suggesting that we may store and process information in an emotion-specific manner.

DeSteno, Petty, Rucker, Wegner, and Braverman (2000) found evidence of selective processing in persuasion contexts. Participants in their studies were induced to either feel sadness, anger, or neutral feelings. Next, they were exposed to arguments for a tax increase that suggested either that sad or angering events would occur if the tax were not supported. Whereas sad participants found the sad arguments most compelling, angry participants were most convinced by the angry arguments. Again it appears that people are particularly sensitive to emotional stimuli that reflect their own emotional states. Angry people do not find sad messages convincing; they find angry messages convincing.

Not only can anger cause selective attention to anger-congruent stimuli, but, consistent with the ATF, selective attention to certain features of a situation can themselves cause anger. In one of their studies, Lerner and Keltner (2001) found that manipulated controllability appraisals mediated the relationship between anger and optimistic risk estimates. Another study found that individuals' lacking of perspective-taking related to their propensity to become angry (Mohr, Howells, Gerace, Day, & Wharton, 2007). Specifically, those low in trait perspective-taking experienced an increase in anger following a personal provocation. Consistent with the bidirectional causality predicted by the ATF, a lack of systematic processing can serve as a cause or an effect of anger.

17.3.2.2 *Depth-of-Processing Effects*

Researchers who have questioned the extent to which emotions may trigger deep versus shallow thought have found anger to be a special case. Early investigations of this question focused on the effects of positive affect, as compared to neutral states, and found that positive affect increased creativity, breadth of thought, and flexibility in ideas (Fredrickson, 2001; Fredrickson & Branigan, 2005; Isen & Geva, 1987). Later, researchers considered the effects of affective states in the context of cognitive processing. In this literature, negative affect (typically operationalized as sadness) was associated with careful processing, whereas positive affect was associated with faster, more spontaneous processing (Bless et al., 1996; Forgas, 1998; Forgas & Fiedler, 1996; Schwarz, Bless, & Bohner, 1991). Forgas (1998) found that happy participants were more likely than sad participants to demonstrate a correspondence bias, overattributing behavior to individual characteristics rather than to situational influences. Similarly, Bodenhausen and colleagues (1994) found that happiness increased reliance on the use of stereotypes (also see Bless et al., 1996).⁶

⁶Such heuristic processing is not always harmful, however. For example, Bless et al. (Bless et al., 1996) have shown that reliance on general knowledge structures is efficient and allows happy participants to succeed at a secondary task because they have processing resources left over.

Yet recent studies suggest that specific emotions, rather than emotional valence, drive depth-of-processing effects. Specifically, participants induced to feel angry have not engaged in the same careful, detailed processing as those induced to feel “negative affect” in previous studies. Tiedens (2001b) found that people induced to feel anger made inferences about others’ motives based on accessible cognitive scripts, whereas people induced to feel sadness seemed to consider more alternatives. Bodenhausen et al. (1994) found that people induced to feel anger engaged in more stereotyping than people induced to feel sadness, and that in persuasion paradigms they could be convinced by relatively superficial characteristics of the speaker (also see Tiedens & Linton, 2001). Small and Lerner (2005) found that participants induced to feel anger chose to provide less public assistance to welfare recipients than those induced to feel sad, unless the sad participants were under cognitive load, in which case sad participants resembled angry participants. Imposing a cognitive-processing constraint on participants changed the choices of sad participants but not those of angry participants.

In these studies, people who felt sad seemed to process stimuli in an effortful and thorough manner. Thus, in the depth-of-processing literature, the effects of anger are quite similar to the effects of happiness (Bodenhausen et al., 1994; Forgas, 1998; Tiedens, 2001b), but entirely different from the effects of other negative emotions, such as sadness.

Consistent with the ATF, Tiedens and Linton (2001) argued that the processing effects of emotional states may be best predicted by understanding the appraisal content of those emotions. Specifically, they suggested that the certainty dimension is more important than the valence dimension in determining whether an emotion results in heuristic or systematic processing. Feeling uncertain has consistently been linked with more systematic processing, just as feeling certain has been linked to more heuristic processing. In a series of studies, Tiedens and Linton (2001) provided evidence that emotions associated with a sense of certainty, such as anger, result in heuristic processing, whereas emotions associated with uncertainty result in systematic processing. Further, they found that certainty appraisals mediate these effects and that when certainty appraisals are manipulated independently from emotion, certainty plays a causal role in determining whether people engage in heuristic or systematic processing. In sum, the ATF can explain how and why anger elicits relatively heuristic processing.

Although anger can lead to decreased depth of processing (Tiedens & Linton, 2001), the impact of this effect on judgment varies. Angry individuals will be more biased than neutral individuals in a judgmental context in which additional mental resources will aid decision-making. However, in some contexts, more thinking can produce worse judgments. For example, the introduction of an arbitrary anchor (e.g., “Is the Mississippi river longer or shorter than 5,000 miles?”) can influence the judged magnitude of that quantity. In one experiment, researchers (Bodenhausen, Gabriel, & Lineberger, 2000) found that sadness, an emotion associated with increased depth of processing, increased reliance on arbitrary anchors in judgment. The decreased depth of processing associated with anger may be a boon in some situations, as the lack of attention to biasing information could attenuate bias.

Young and Tiedens (2009) suggest that there might be some instances when anger actually results in greater processing. Because anger is associated with the desire to confront, oppose, and argue, it may be that when individuals become angry, they become particularly vigilant about creating oppositional arguments. Beyond providing evidence for this process, Young and Tiedens (2008) showed that it can produce normatively better responses. Specifically, since individuals who were angry were more interested in opposing views, they engaged in better hypothesis testing. Compared to participants who had been induced to feel sadness or neutral feeling, participants induced to feel anger avoided the tendency to only examine hypothesis confirming evidence and instead sought out information that could invalidate prior hypotheses regardless of whether these hypotheses were self-generated or provided by the experimenter.

17.4 Possible Mechanisms Underlying Anger Effects Predicted by the ATF

The studies we have reviewed identify the effects of specific emotions in general and the effects of appraisal tendencies in particular on judgment and decision-making. Yet, as with any newly documented phenomena, we do not yet understand the causal mechanisms underlying the unique effects of anger. By contrast, a number of accounts have considered how global moods influence the processing and outcomes of thought, including those emphasizing network associations, informational roles of mood, and motivational roles of mood. However, the specific effects of anger contradict the positive–negative dichotomy that these theories assume, complicating their account of the causal mechanisms underlying the effects of emotions. Yet, when combined with the ATF, these accounts could help to explain the unique anger’s effects, including heuristic processing and optimistic perceptions of risk.

17.4.1 *Associative Network Mechanisms*

Research on mood congruency has long suggested that people have “affective associative networks,” meaning that thoughts that create positive affect are stored close to one another in memory, as are thoughts that create negative affect (Bower, 1981, 1991; Forgas, 1995). Some researchers in this tradition have argued that these associated networks are best characterized at the specific-emotion level rather than at the global-affect level (Halberstadt & Niedenthal, 1997; Niedenthal, Halberstadt, & Innes-Ker, 1999). Taking this reasoning a step further, according to the ATF, nodes in associative networks may be linked by appraisal themes. If so, mood-congruent attention, priming, and retrieval effects should occur not just between an emotional state and stimuli connected to that emotional state, but between an emotional state and stimuli connected to its central appraisals. For example, fearful people facing an uncertain situation may have a low-control, low-certainty, low-coping potential network activated, thus reminding them of past situations where they felt helpless and unsure. Because angry people facing the same uncertain situation may have a high-control, high-certainty, high-coping potential network activated instead, what is salient to them will be quite different. For example, they might focus on what they can do to alter the situation, or who might be responsible for causing their aggravation. These salient memories and sensitivities may play an important role in determining how individuals form risk estimates, assign causality and blame, and form optimistic self-perceptions.

17.4.2 *Informational Mechanisms*

Another possibility that has been explored is the idea that people’s emotional states directly inform their judgment (Schwarz & Clore, 1983; Slovic, Finucane, Peters, & MacGregor, 2002). These informational approaches argue that people sometimes overgeneralize the valence of an emotional state when deciding whether a situation is benign or potentially problematic, like when someone glum about the weather decides that their life is similarly bleak (Schwarz & Clore, 1983). The appraisal content of specific emotions also offers information that people may overgeneralize to subsequent novel situations, thus influencing their future judgments and decisions (Schwarz, 2002). In this framework, the lingering appraisal of uncertainty that accompanies fear would be used as information about the nature of a subsequent risky choice. Appraisals associated with the emotional state, such as whether a situation is certain or uncertain, situational or controlled by oneself or

others, become specific information about the nature of the judgment or decision itself. Thus, unlike the associative network framework where the carryover effect is explained by spreading activation, according to this framework, an informational mechanism explanation posits that in some sense the relevance of an appraisal for a subsequent judgment is inferred.

17.4.3 Motivational Mechanisms

Many researchers have speculated that once a mood affects judgment, it can also activate a motivation that influences both judgment outcomes and processing. For example, an individual might become risk seeking in an attempt to ameliorate her sad mood (Raghunathan & Pham, 1999). Along these lines, negative moods have been associated with a “mood repair” motive, while positive moods have been associated with a “mood maintenance” motive (Isen & Geva, 1987; Isen, Nygren, & Ashby, 1988). It is hard to see how an optimistic bias might be motivated by an attempt to remain angry. While an angry state could be sustained through (optimistic) rumination, it would require that these individuals find the angry state desirable. Thus, although anger may be, in many respects, a positive emotion (see below), mood maintenance theories do not offer compelling predictions for anger.

Aside from the human motivation to “feel good,” the ATF could identify other motivations that might explain the effects of anger on the thought processes and outcomes. For example, anger has been associated with appraisals of injustice (Lazarus, 1991a), and particularly with violations of individual rights (Rozin, Lowery, Imada, & Haidt, 1999). Given that perceived injustice often creates the motivation to restore justice (Solomon, 1990), angry people’s judgments of criminals and unjust behaviors are likely to be particularly harsh (Goldberg et al., 1999; Lerner et al., 1998). In situations in which they believe that greater cognitive processing would redress injustice, angry people may actually process more than sad people, effectively reversing the tendency for angry people to process more heuristically than sad people.

While research on the effects of positive and negative mood may have obscured important sources of variation among specific emotions, this literature has successfully identified mechanisms through which affect influences judgment and decision-making. As researchers begin to focus on the effects of specific emotions such as anger, these previous investigations can serve a model, while also becoming more exact by accounting for appraisal tendencies. Finally, the possibility that specific emotions generate emotion-specific mechanisms deserves research attention as well.

17.5 Is Anger a Negative or a Positive Emotion?

Decades of emotion research and theory have classified anger as a negative emotion (for reviews, see Ben-Ze-ev, 2000; Berkowitz & Harmon-Jones, 2004; Lazarus, 1991a). Yet, as indicated by the findings we have presented, anger does not follow many of the typical patterns associated with negative emotions. Rather than triggering pessimism, it triggers optimism about one’s own outcomes (Fischhoff et al., 2005; Hemenover & Zhang, 2004; Lerner et al., 2003; Lerner & Keltner, 2000, 2001). It prompts careless thought, not careful thought (Bodenhausen et al., 1994; Lerner et al., 1998; Small & Lerner, 2005; Tiedens, 2001b; Tiedens & Linton, 2001). Rather than focusing attention on all negative events, it focuses attention only on angering events (DeSteno et al., 2004; DeSteno, Petty, Rucker, Wegener, & Braverman, 2004). Some researchers have argued that anger even resembles happiness in terms of hemispheric laterality (Harmon-Jones & Sigelman, 2001; Harmon-Jones et al.,

2003, Harmon-Jones et al., this book). Historically, the left frontal cortical region of the brain has corresponded not only with approach motivation but also with positive affective processes, whereas the right frontal cortical region has corresponded with withdrawal motivation and negative affective processes (for reviews, see Coan & Allen, 2003; R. J. Davidson, 1995; Richard J. Davidson, Jackson, & Kalin, 2000; Fox, 1991; Silberman & Weingartner, 1986). By contrast, both state and trait anger are associated with relatively greater *left* frontal cortical activity than right frontal activity (for a review, see Harmon-Jones et al., this book).

With this evidence in mind, one might wonder whether anger is actually a positive emotion, a proposition consistent with some of the earliest scholarly work on anger. Aristotle's *Rhetoric* (350 BCE/1991), for example, elucidates several seemingly positive consequences of anger, including optimism about attaining one's goals and a pleasurable anticipation of vengeance:

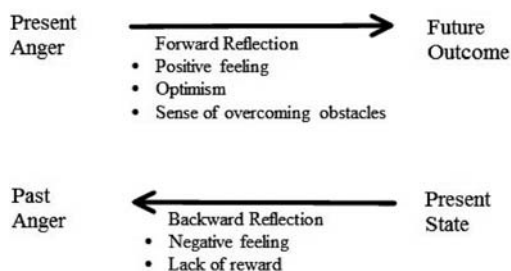
For since nobody aims at what he thinks he cannot attain, the angry man is aiming at what he can attain, and the belief that you will attain your aim is pleasant. Hence it has been well said about wrath, 'Sweeter it is by far than the honeycomb dripping with sweetness, And spreads through the hearts of men.'

It is also attended by a certain pleasure because the thoughts dwell upon the act of vengeance, and the images then called up cause pleasure, like the images called up in dreams (p. 146).

To reconcile the question of whether anger is a positive or negative emotion, we propose assessing the potential positivity of anger from a temporal perspective. Specifically, we propose that anger will be experienced as relatively unpleasant and unrewarding when one is reflecting back on the source of the anger but may be experienced as relatively pleasant and rewarding when one is looking toward the future.

Consider that an event that elicits anger typically involves someone blocking your goals or offending you or someone close to you. Based on past research, we argue that unpleasantness and lack of reward characterize anger at this stage (see Fig. 17.1). Studies that have asked people to recall emotional events from their lives have found that people rate the events that triggered their anger as both negative and unpleasant (Carlsmith, Wilson, & Gilbert, 2007; Ellsworth & Smith, 1988; C. A. Smith & Ellsworth, 1985). Similarly, studies that induced anger in the laboratory have found that participants rate their feelings in response to the anger induction as negative and unpleasant (Gross & Levenson, 1995). By contrast, studies have found that people experience and recall events that trigger happiness as quite pleasant (Ellsworth & Smith, 1988; C. A. Smith & Ellsworth, 1985).⁷ In this sense, anger differs from happiness and resembles other "negative" emotions, such as fear and sadness.

Fig. 17.1 Anger in forward and backward reflection



⁷There may be exceptions to the overall pattern of negativity in backward reflection. For example, Parrott (1993) has written about the phenomenon of "storming around." There may be enjoyment to be gained from dwelling on how one has been wronged, but the experience is generally unpleasant.

Now consider that when engaging in forward reflection, an angry person considers not the original emotion-triggering event, but instead possible future actions. Implicitly or explicitly, the angry person formulates a plan to address the source of the anger or to address new goals. For an angry person, we argue, such forward reflection can be pleasant and rewarding. In support of this view, consider the appraisal and action tendencies associated with anger: a belief that one can control and improve a situation, and an expectation of conquering opponents and obstacles (Frijda et al., 1989). In addition, research has found that angry people tend to believe they will get what they want across multiple domains, including health, social relations, career, social competence, and political concerns (Lerner et al., 2003; Lerner & Keltner, 2000, 2001).

Interestingly, the forward-reflection stage of anger triggers not only a positive outlook but also a positive sense of self. Studies have found that angry people often feel “more energized” to assault the cause of their anger (Frijda et al., 1989). According to Shaver et al. (1987 p. 1078), “. . . the angry person reports becoming stronger (higher in potency) and more energized in order to fight or rail against the cause of anger.” Anger may be especially exhilarating when one is anticipating revenge or punishment (Carlsmith et al., 2007; de Quervain et al., 2004; Knutson, 2004; Tripp & Bies, 1997) or witnessing the misfortune of disliked others (Leach, Spears, Branscombe, & Doosje, 2003; R. H. Smith et al., 1996). Moreover, recent research has also showed that individuals can prefer to be in an angry state in order to succeed at a confrontational task where being angry would be useful (Tamir, Mitchell, & Gross, *in press*). Reflecting on his childhood memory of watching the Germans fail to win gold medals in the 1936 Olympics, for example, historian Peter Gay described *Schadenfreude* as “one of the great joys of life” (Rothstein, 2000).

Brain-imaging studies are beginning to reveal the neural systems that underlie such joyful wrath. Some neuroeconomists argue that the dorsal striatum – a sub-cortical brain structure activated when one anticipates punishing a transgressor – is associated with reward and thus provides evidence for the pleasurable nature of punishment (de Quervain et al., 2004; Knutson, 2004). Moreover, the striatum remains activated even if administering punishment comes at a personal cost. In this case, the medial prefrontal cortex also becomes activated, presumably in the service of balancing costs and benefits (de Quervain et al., 2004; Knutson, 2004). Such imaging studies could test the hypothesis that reward centers of the brain will become differentially engaged as a function of forward or backward reflection.

Other neuroscientific lines of inquiry may also help to distinguish between backward and forward reflection. Notably, Harmon-Jones and colleagues (2003) have found that the relationship between anger and left frontal cortical activity appears only in situations where one has an opportunity to approach the source of the anger. Anger may not trigger the same hemispheric pattern as happiness when there is no opportunity to approach because the situation facilitates only backward reflection.

In describing the anticipatory pleasure of anger, we are not arguing that the emotion is associated with purely positive outcomes. On the contrary, the highly pleasurable exhilaration associated with anger may portend a significant fall. Perhaps like heroin and other addictive substances, anger may be rewarding in the anticipation and experiential stages (see Ainslie, 2003) but harmful in the long run. The “rush” and optimism associated with anger may lead people to make unwise choices that overlook their own abilities, their interdependence on others, social norms, and other goals. Thus, the positive aspects of anger could lay the groundwork for some of its very negative consequences, such as violence and aggression.

It is also important to keep in mind the role of individual differences. We have sketched hypotheses for two main processes: forward and backward reflection. In both cases, however, individual differences may color the overall pleasantness of anger. For example, individuals who are high on trait anger regard the experience of anger less negatively than do individuals low on trait anger (Harmon-Jones, 2004). This may be because the experience of chronic anger (lacking some specific,

unpleasant trigger) is actually rewarding in some way. It is not known, however, whether the ratings of high-trait-anger individuals would correspond to positive experience in an absolute sense or merely in a relative sense (i.e., compared to ratings of low-trait-anger individuals). For example, do high-trait-anger individuals experience state anger to be as pleasurable as the experience of state happiness?

Gender may also be an important determinant of how anger is felt. For example, Campbell and Muncer's (1994) finding that men typically see the expression of anger as seizing control of the situation and exerting dominance while women more typically view the expression of anger as a loss of self-control has been replicated cross-nationally (Archer & Haigh, 1999; Ramirez, Andreu, & Fujihara, 2001; Richardson & Huguet, 2001). One current hypothesis is that women are more reluctant to express anger and do so only at higher intensities, which is when they are more likely to feel they have lost control (e.g., Astin, Redston, & Campbell, 2003). Are women thus more likely to experience anger as a negative emotion?

In any event, the ATF suggests that if an individual is feeling a mix of anger and sadness, the effects of this emotional state will be determined by the mix of appraisals experienced. If an instance of sadness and anger is characterized by high certainty and situational control, we would expect an individual to carry those appraisals over to new situations. When making judgments about control of future situations, we would expect this individual to respond like a typical sad person; when making judgments involving certainty, we would expect this person to respond more like a typical angry person. As another example, consider situations in which people typically feel a mixture of happiness and sadness, as when a parent watches a child go off to college. Some recent data suggest that such a mixed state results in increased cognitive processing (Fong, 2006) perhaps due to the sense of uncertainty that accompanies this ambivalent state. In other words, it may be that emotional ambivalence, although a mix of two emotions, may have an appraisal profile distinct from its constituent emotions. But, just like any other emotional state, we believe that this emotional profile will affect the judgments and processing of those who experience it. The ATF therefore is well-suited for the study of mixed emotions, a topic that merits further examination.

17.6 Conclusion

A unique and complex emotion, anger cannot reasonably be clustered with other negative emotions when making predictions about human judgments and decisions. Angry decision makers feel negatively about past events, yet also make optimistic predictions of their likelihood of success in a variety of life domains (Fischhoff et al., 2005; Lerner et al., 2003; Lerner & Keltner, 2000, 2001). This optimism derives primarily from a sense of certainty and predictability, as well as from a sense of control over outcomes (Lerner & Keltner, 2001). Angry decision makers rely on heuristics when processing information, not stopping to ponder alternative options before acting (Bodenhausen et al., 1994; Lerner et al., 1998; Small & Lerner, 2005; Tiedens, 2001b; Tiedens & Linton, 2001). This tendency also derives primarily from the sense of certainty associated with anger and perhaps from the optimism angry decision makers have about the future.

As Aristotle wrote, angry decision makers may have a difficult time being angry at the right time, for the right purpose, and in the right way. Their emotional experiences and appraisals may hinder their ability to view a situation objectively and rationally. Instead, they approach situations with confidence, a sense of control, and negative thoughts about others. In some situations, these appraisal tendencies may cascade into undesirable outcomes, such as aggression, unrealistic optimism, and overconfidence. Yet these tendencies can also cascade into desirable outcomes, as when anger buffers

decision makers from indecision, risk aversion, and overanalysis. The many judgment and decision outcomes associated with anger must be documented and their normative status in diverse situations evaluated.

Acknowledgment Grants from the National Institute of Mental Health (MH62376) and the National Science Foundation (PECASE SES0239637) supported this project. We thank the Center for Public Leadership at the Harvard Kennedy School of Government for administrative support. We also thank Max Bazerman for his countless acts of support.

References

- Ainslie, G. (2003). Uncertainty as wealth. *Behavioural Processes*, 64, 369–385.
- Archer, J., & Haigh, A. (1999). Sex differences in beliefs about aggression: Opponent's sex and the form of aggression. *British Journal of Social Psychology*, 38, 71–84.
- Aristotle (350 BCE/1991) *Rhetoric* (H. Lawson-Tancred, Trans.). New York: Oxford University Press.
- Astin, S., Redston, P., & Campbell, A. (2003). Sex differences in social representations of aggression: Men justify, women excuse? *Aggressive Behavior*, 29, 128–133.
- Averill, J. R. (1982). *Anger and aggression: An essay on emotion*. New York: Springer-Verlag.
- Ben-Ze-ev, A. (2000). *The subtlety of emotions*. Cambridge, MA: MIT Press.
- Berkowitz, L. (1990). On the formation and regulation of anger and aggression: A cognitive-neoassociationistic analysis. [Journal]. *American Psychologist*, 45(4), 494–503.
- Berkowitz, L., & Harmon-Jones, E. (2004). Toward an understanding of the determinants of anger. *Emotion*, 4(2), 107–130.
- Bless, H., Clore, G. L., Schwarz, N., Golisano, V., Rabe, C., & Wolk, M. (1996). Mood and the use of scripts: Does a happy mood really lead to mindlessness? *Journal of Personality and Social Psychology*, 71(4), 665–679.
- Bodenhausen, G. V. (1993). Emotions, arousal, and stereotypic judgments: A heuristic model of affect and stereotyping. In D. M. Mackie, & D. L. Hamilton (Eds.), *Affect, cognition and stereotyping: Interactive processes in group perception* (pp. 13–37). San Diego, CA: Academic Press.
- Bodenhausen, G. V., Gabriel, S., & Lineberger, M. (2000). Sadness and susceptibility to judgmental bias: The case of anchoring. [Journal]. *Psychological Science*, 11(4), 320–323.
- Bodenhausen, G. V., Kramer, G. P., & Süsser, K. (1994). Happiness and stereotypic thinking in social judgment. [Journal]. *Journal of Personality and Social Psychology*, 66(4), 621–632.
- Bodenhausen, G. V., Sheppard, L. A., & Kramer, G. P. (1994). Negative affect and social judgment: The differential impact of anger and sadness. [Journal. Special Issue: Affect in Social Judgments and Cognition]. *European Journal of Social Psychology*, 24(1), 45–62.
- Bower, G. H. (1981). Mood and memory. *American Psychologist*, 36(2), 129–148.
- Bower, G. H. (1991). Mood congruity of social judgment. In J. Forgas (Ed.), *Emotion and social judgment* (pp. 31–54). Oxford, England: Pergamon Press.
- Cacioppo, J. T., Priester, J. R., & Berntson, G. G. (1993). Rudimentary determinants of attitudes. II: Arm flexion and extension have differential effects on attitudes. *Journal of Personality and Social Psychology*, 65, 5–17.
- Camerer, C. (2001). *Social preferences in dictator, ultimatum, and trust games behavioral game theory: Experiments on strategic interaction*. Princeton: Princeton University Press.
- Campbell, A., & Muncer, S. (1994). Sex differences in aggression: social representation and social roles. *British Journal of Social Psychology*, 33, 233–240.
- Carlsmith, K., Wilson, T. D., & Gilbert, D. (2007). The Paradoxical Consequences of Revenge. *Manuscript under review*.
- Caspi, A., Elder, G. H., & Bem, D. J. (1987). Moving against the world: Life-course patterns of explosive children. *Developmental Psychology*, 23(2), 308–313.
- Clark, M. S., Pataki, S. P., & Carver, V. H. (1996). Some thoughts and findings on self-presentation of emotions in relationships. In G. J. O. Fletcher, & J. Fitness (Eds.), *Knowledge structures in close relationships: A social psychological approach* (pp. 247–274). Hillsdale, NJ: Lawrence Erlbaum Associates.
- Clore, G. L. (1994). Why emotions require cognition. In P. Ekman, & R. J. Davidson (Eds.), *The nature of emotion: Fundamental questions* (pp. 181–191). New York: Oxford University Press.
- Coan, J. A., & Allen, J. J. B. (2003). The state and trait of frontal EEG activation asymmetries over the frontal cortex. In K. Hugdahl, & R. J. Davidson (Eds.), *The asymmetrical brain* (pp. 565–615). Cambridge, MA: MIT Press.

- Cohen, D., Nisbett, R. E., Bowdle, B. F., & Schwarz, N. (1996). Insult, aggression, and the southern culture of honor: An "experimental ethnography". *Journal of Personality and Social Psychology*, 70, 945–960.
- Davidson, R. J. (1995). Cerebral asymmetry, emotion, and affective style. In R. J. Davidson, & K. Hugdahl (Eds.), *Brain asymmetry* (pp. 361–387). Cambridge, MA: MIT Press.
- Davidson, R. J., Jackson, D. C., & Kalin, N. H. (2000). Emotion, plasticity, context, and regulation: Perspectives from affective neuroscience. *Psychological Bulletin*, 126(6), 890–909.
- de Quervain, D. J. -F., Fischbacher, U., Treyer, V., Schellhammer, M., Schnyder, U., Buck, A., et al. (2004). The neural basis of altruistic punishment. *Science*, 305, 1254–1258.
- DeSteno, D., Dasgupta, N., Bartlett, M. Y., & Caidric, A. (2004). Prejudice from thin air: The effect of emotion on automatic intergroup attitudes. *Psychological Science*, 15(5), 319–324.
- DeSteno, D., Petty, R. E., Rucker, D. D., Wegener, D. T., & Braverman, J. (2004). Discrete emotions and persuasion: The role of emotion-induced expectancies. *Journal of Personality and Social Psychology*, 86(1), 43–56.
- DeSteno, D., Petty, R. E., Wegener, D. T., & Rucker, D. D. (2000). Beyond valence in the perception of likelihood: The role of emotion specificity. *Journal of Personality and Social Psychology*, 78(3), 397–416.
- Dunn, J. R., & Schweitzer, M. E. (2005). Feeling and believing: The influence of emotion on trust. *Journal of Personality and Social Psychology*, 88(5), 736–748.
- Ekman, P., Levenson, R. W., & Friesen, W. V. (1983). Autonomic nervous system activity distinguishes among emotions. *Science*, 221(4616), 1208–1210.
- Ellsworth, P. C., & Scherer, K. R. (2003). Appraisal processes in emotion. In R. J. Davidson, K. R. Scherer, & H. H. Goldsmith (Eds.), *Handbook of affective sciences* (pp. 572–595). New York: Oxford University Press.
- Ellsworth, P. C., & Smith, C. A. (1988). From appraisal to emotion: Differences among unpleasant feelings. *Motivation and Emotion*, 12(3), 271–302.
- Fischhoff, B., Gonzalez, R. M., Lerner, J. S., & Small, D. A. (2005). Evolving judgments of terror risks: Foresight, hindsight, and emotion. [Manuscript under review]. *Journal of Experimental Psychology: Applied*, 11(2), 124–139.
- Fong, C. T. (2006). The effects of emotional ambivalence on creativity. *Academy of Management Journal*, 49(5), 1016–1030.
- Forgas, J. P. (1995). Mood and judgment: The affect infusion model (AIM). *Psychological Bulletin*, 117(1), 39–66.
- Forgas, J. P. (1998). On being happy and mistaken: Mood effects on the fundamental attribution error. *Journal of Personality and Social Psychology*, 75, 318–331.
- Forgas, J. P. (2003). Affective influences on attitudes and judgments. In R. J. Davidson, K. R. Scherer, & H. H. Goldsmith (Eds.), *Handbook of affective science* (pp. 596–618). New York: Oxford University Press.
- Forgas, J. P., & Fiedler, K. (1996). Us and them: Mood effects on intergroup discrimination. *Journal of Personality and Social Psychology*, 70(1), 28–40.
- Fox, N. A. (1991). If it's not left, it's right: Electroencephalograph asymmetry and the development of emotion. *American Psychologist*, 46(8), 863–872.
- Fredrickson, B. L. (2001). The role of positive emotions in positive psychology. The broaden-and-build theory of positive emotions. *American Psychologist*, 56(3), 218–226.
- Fredrickson, B. L., & Branigan, C. (2005). Positive emotions broaden the scope of attention and thought-action repertoires. *Cognition and Emotion*, 19, 313–332.
- Fredrickson, B. L., Maynard, K. E., Helms, M. J., Haney, T. L., Siegler, I. C., & Barefoot, J. C. (2000). Hostility predicts magnitude and duration of blood pressure response to anger. *Journal of Behavioral Medicine*, 23(3), 229–243.
- Frijda, N. H. (1986). *The emotions*. New York: Cambridge University Press.
- Frijda, N. H. (1988). The laws of emotion. *American Psychologist*, 43, 349–358.
- Frijda, N. H. (1994). Emotions require cognitions, even if simple ones. In P. Ekman, & R. J. Davidson (Eds.), *The nature of emotion: Fundamental questions* (pp. 197–202). New York: Oxford University Press.
- Frijda, N. H., Kuipers, P., & ter Schure, E. (1989). Relations among emotion, appraisal, and emotional action readiness. *Journal of Personality and Social Psychology*, 57, 212–228.
- Goldberg, J. H., Lerner, J. S., & Tetlock, P. E. (1999). Rage and reason: the psychology of the intuitive prosecutor. *European Journal of Social Psychology*, 29(5-6), 781–795.
- Gross, J. J., & Levenson, R. W. (1995). Emotion elicitation using films. [Journal]. *Cognition and Emotion*, 9(1), 87–108.
- Guth, W., Schmittberger, R., & Schwarze, B. (1982). An experimental analysis of ultimatum bargaining. *Journal of Economic Behavior & Organization*, 3(4), 367–388.
- Halberstadt, J. B., & Niedenthal, P. M. (1997). Emotional state and the use of stimulus dimensions in judgment. *Journal of Personality and Social Psychology*, 72(5), 1017–1033.
- Hansen, C. H., & Hansen, R. D. (1988). Finding the face in the crowd: An anger superiority effect. *Journal of Personality and Social Psychology*, 54(6), 917–924.

- Harmon-Jones, E. (2004). On the relationship of frontal brain activity and anger: Examining the role of attitude toward anger. *Cognition, & Emotion*, 18(3), 337–361.
- Harmon-Jones, E. (2007). *Trait anger predicts relative left frontal cortical activation to anger-inducing stimuli. International Journal of Psychophysiology, In Press, Corrected Proof.*
- Harmon-Jones, E., & Sigelman, J. (2001). State anger and prefrontal brain activity: Evidence that insult-related relative left-prefrontal activation is associated with experienced anger and aggression. *Journal of Personality and Social Psychology*, 80(5), 797–803.
- Harmon-Jones, E., Sigelman, J., Bohlig, A., & Harmon-Jones, C. (2003). Anger, coping, and frontal cortical activity: The effects of coping potential on anger-induced left frontal activity. *Cognition, & Emotion*, 17(1), 1–24.
- Haviland, J. M., & Lelwica, M. (1987). The induced affect response: 10-week-old infants' responses to three emotion expressions. *Developmental Psychology*, 23(1), 97–104.
- Hemenover, S. H., & Zhang, S. (2004). Anger, personality and optimistic stress appraisals. *Cognition, & Emotion*, 18(3), 363–382.
- Hochschild, A. (1983). *The managed heart: The commercialization of human feeling*. Berkeley, CA: University of California Press.
- Isen, A. M., & Geva, N. (1987). The influence of positive affect on acceptable level of risk: The person with a large canoe has a large worry. *Organizational Behavior and Human Decision Processes*, 39(2), 145–154.
- Isen, A. M., Nygren, T. E., & Ashby, F. G. (1988). Influence of positive affect on the subjective utility of gains and losses: It is just not worth the risk. *Journal of Personality and Social Psychology*, 55(5), 710–717.
- Isen, A. M., Shalke, T. E., Clark, M., & Karp, L. (1978). Affect, accessibility of material in memory, and behavior: A cognitive loop? *Journal of Personality and Social Psychology*, 36, 1–12.
- Johnson, E. J., & Tversky, A. (1983). Affect, generalization, and the perception of risk. *Journal of Personality and Social Psychology*, 45(1), 20–31.
- Johnson-Laird, P. N., & Oatley, K. (1992). Basic emotions, rationality, and folk theory. *Cognition, & Emotion*, 6(3–4), 201–223.
- Kahneman, D., & Lovallo, D. (1993). Timid choices and bold forecasts: A cognitive perspective on risk taking. *Management Science*, 39(1), 17–31.
- Keltner, D., Ellsworth, P. C., & Edwards, K. (1993). Beyond simple pessimism: Effects of sadness and anger on social perception. *Journal of Personality and Social Psychology*, 64(5), 740–752.
- Keltner, D., & Gross, J. J. (1999). Functional accounts of emotions. *Cognition, & Emotion*, 13(5), 467–480.
- Keltner, D., Locke, K. D., & Audrain, P. C. (1993). The influence of attributions on the relevance of negative feelings to personal satisfaction. *Personality and Social Psychology Bulletin*, 19(1), 21–29.
- Knoch, D., Pascual-Leone, A., Meyer, K., Treyer, V., & Fehr, E. (2006). Diminishing reciprocal fairness by disrupting the right prefrontal cortex. *Science*, 314(5800), 829–832.
- Knutson, B. (2004). Sweet revenge? [Magazine]. *Science*, 305, 1246–1247.
- Lazarus, R. S. (1991a). *Emotion and adaptation*. New York: Oxford University Press.
- Lazarus, R. S. (1991b). Progress on a cognitive-motivational-relational theory of emotion. *American Psychologist*, 46(8), 819–834.
- Lazarus, R. S. (1994). Appraisal: The long and short of it. In P. Ekman, & R. J. Davidson (Eds.), *The nature of emotion: Fundamental questions* (pp. 208–215). London: Oxford University Press.
- Leach, C. W., Spears, R., Branscombe, N. R., & Doosje, B. (2003). Malicious pleasure: Schadenfreude at the suffering of another group. *Journal of Personality and Social Psychology*, 84, 932–943.
- Lejuez, C. W., Read, J. P., Kahler, C. W., Richards, J. B., Ramsey, S. E., & Stuart, G. L. (2002). Evaluation of a behavioral measure of risk taking: The balloon analogue risk task (BART). *Journal of Experimental Psychology: Applied*, 8(2), 75–84.
- Lerner, J. S., Dahl, R., Hariri, A. R., & Taylor, S. (2007). Facial expressions of emotion reveal neuroendocrine and cardiovascular stress responses. *Biological Psychiatry*, 61, 253–260.
- Lerner, J. S., Goldberg, J. H., & Tetlock, P. E. (1998). Sober second thought: The effects of accountability, anger and authoritarianism on attributions of responsibility. *Personality and Social Psychology Bulletin*, 24(6), 563–574.
- Lerner, J. S., Gonzalez, R. M., Small, D. A., & Fischhoff, B. (2003). Effects of fear and anger on perceived risks of terrorism: A national field experiment. *Psychological Science*, 14(2), 144–150.
- Lerner, J. S., & Keltner, D. (2000). Beyond valence: Toward a model of emotion-specific influences on judgment and choice. *Cognition and Emotion*, 14(4), 473–493.
- Lerner, J. S., & Keltner, D. (2001). Fear, anger, and risk. *Journal of Personality and Social Psychology*, 81(1), 146–159.
- Lerner, J. S., Small, D. A., & Loewenstein, G. (2004). Heart strings and purse strings: Effects of emotions on economic transactions. *Psychological Science*, 15, 337–341.
- Laner, J. S., & Tiedens, L. Z. (2006). Portrait of the angry decision maker: how appraisal tendencies anger's influence on cognition. *Journal of Behavioral Decision Making*, 19(2), 115–137.

- Levenson, R. W. (1994). Human emotion: A functional view. In P. Ekman, & R. J. Davidson (Eds.), *The nature of emotion: Fundamental questions* (pp. 123–126). New York: Oxford University Press.
- Loewenstein, G., & Lerner, J. S. (2003). The role of affect in decision making. In R. Davidson, K. Scherer, & H. Goldsmith (Eds.), *Handbook of affective science* (pp. 619–642). London: Oxford University Press.
- Mackie, D. M., Devos, T., & Smith, E. R. (2000). Intergroup emotions: Explaining offensive action tendencies in an intergroup context. *Journal of Personality and Social Psychology*, 79(4), 602–616.
- Mayer, J. D., & Hanson, E. (1995). Mood-congruent judgment over time. *Personality and Social Psychology Bulletin*, 21(3), 237–244.
- McDaniels, T. L., Axelrod, L. J., Cavanagh, N. S., & Slovic, P. (1997). Perception of ecological risk to water environments. *Risk Analysis*, 17(3), 341–352.
- Mohr, P., Howells, K., Gerace, A., Day, A., & Wharton, M. (2007). The role of perspective taking in anger arousal. *Personality and Individual Differences*, 43(3), 507–517.
- Musch, J., & Klauer, K. C. (2004). *The psychology of evaluation: affective processes in cognition and emotion*. Mahwah, NJ: Lawrence Erlbaum.
- Neumann, R. (2000). The causal influences of attributions on emotions: A procedural priming approach. *Psychological Science*, 11(3), 179–182.
- Niedenthal, P. M., Halberstadt, J. B., & Innes-Ker, A. H. (1999). Emotional response categorization. *Psychological Review*, 106(2), 337–361.
- Niedenthal, P. M., Halberstadt, J. B., & Setterlund, M. B. (1997). Being happy and seeing “happy”: Emotional state mediates visual word recognition. *Cognition, & Emotion*, 11(4), 403–432.
- Oatley, K., & Jenkins, J. M. (1992). Human emotions: Function and dysfunction. *Annual Review of Psychology*, 43, 55–85.
- Ortony, A., Clore, G. L., & Collins, A. (1988). *The cognitive structure of emotions*. New York: Cambridge University Press.
- Parkinson, B. (1996). Emotions are social. *British Journal of Psychology*, 87(4), 663–683.
- Parrott, W. G. (1993). Beyond hedonism: Motives for inhibiting good moods and for maintaining bad moods. In D. M. Wegner, & J. W. Pennebaker (Eds.), *Handbook of mental control* (pp. 278–305). New York: Prentice Hall.
- Pillutla, M. M., & Murnighan, J. K. (1996). Unfairness, anger, and spite: Emotional rejections of ultimatum offers. *Organizational Behavior and Human Decision Processes*, 68(3), 208–224.
- Plutchik, R. (1979). *A psychoevolutionary synthesis*. New York: Addison-Wesley.
- Quigley, B. M., & Tedeschi, J. T. (1996). Mediating effects of blame attributions on feelings of anger. *Personality and Social Psychology Bulletin*, 22(12), 1280–1288.
- Rabin, M. (1993). Incorporating fairness into game theory and economics. *The American Economic Review*, 83(5), 1281–1302.
- Raghunathan, R., & Pham, M. T. (1999). All negative moods are not equal: Motivational influences of anxiety and sadness on decision making. *Organizational Behavior and Human Decision Processes*, 79(0), 56–77.
- Ramirez, J. M., Andreu, J. M., & Fujihara, T. (2001). Cultural and sex differences in aggression: A comparison between Japanese and Spanish students using two different inventories. *Aggressive Behavior*, 27, 313–322.
- Richardson, D. S., & Huguet, P. (2001). Beliefs about and experience with aggression in the United States and France. In J. M. Ramirez, & D. S. Richardson (Eds.), *Cross-cultural approaches to aggression and reconciliation* (pp. 73–85). New York: Nova Science.
- Roseman, I. J. (1984). Cognitive determinants of emotion: A structural theory. *Review of Personality and Social Psychology*, 5, 11–36.
- Roseman, I. J. (1991). Appraisal determinants of discrete emotions. *Cognition and Emotion*, 5, 161–200.
- Rothstein, E. (2000, February 5). Missing the fun of a minor sin. *The New York Times*, p. B9.
- Rozin, P., Lowery, L., Imada, S., & Haidt, J. (1999). The CAD triad hypothesis a mapping between three moral emotions (contempt, anger, disgust) and three moral codes (community, autonomy, divinity). *Journal of Personality and Social Psychology*, 76(4), 574–586.
- Sanfey, A. G., Rilling, J. K., Aronson, J. A., Nystrom, L. E., & Cohen, J. D. (2003). The neural basis of economic decision-making in the ultimatum game. *Science*, 300, 1755–1758.
- Scherer, K. R. (1999). Appraisal theory. In T. Dalgleish, & M. J. Power (Eds.), *Handbook of cognition and emotion* (pp. 637–663). Chichester, England: John Wiley, & Sons.
- Scherer, K. R. (2001). Appraisal considered as process of multi-level sequential checking. In K. R. Scherer, A. Schorr, & T. Johnstone (Eds.), *Appraisal processes in emotion: Theory, methods, research* (pp. 92–120). New York: Oxford University Press.
- Schwarz, N. (1990). Feelings as information: Informational and motivational functions of affective states. In E. T. Higgins, & R. M. Sorrentino (Eds.), *Handbook of motivation and cognition: Foundations of social behavior* (Vol. 2, pp. 527–561). New York: Guilford Press.

- Schwarz, N. (2002). Feelings as information: Moods influence judgment and processing strategies. In T. Gilovich, D. Griffin, & D. Kahneman (Eds.), *Heuristics and biases: The psychology of intuitive judgment* (pp. 534–547). New York: Cambridge University Press.
- Schwarz, N., Bless, H., & Bohner, G. (1991). Mood and persuasion: Affective states influence the processing of persuasive communications. In M. Zhanna (Ed.), *Advances in experimental social Psychology* (Vol. 24, pp. 161–199). San Diego: Academic Press.
- Schwarz, N., & Clore, G. L. (1983). Mood, misattribution and judgments of well-being: Informative and directive functions of affective states. *Journal of Personality and Social Psychology*, 45(3), 513–523.
- Shaver, P., Schwartz, J., Kirson, D., & O'Connor, C. (1987). Emotion knowledge: Further exploration of a prototype approach. *Journal of Personality and Social Psychology*, 52(6), 1061–1086.
- Siegmán, A. W., & Smith, T. W. (Eds.). (1994). *Anger, hostility, and the heart*. Hillsdale, NJ: Lawrence Erlbaum.
- Silberman, E. K., & Weingartner, H. (1986). Hemispheric lateralization of functions related to emotion. *Brain and Cognition*, 5(3), 322–353.
- Simon, H. A. (1967). Motivational and emotional controls of cognition. *Psychological Review*, 74(1), 29–39.
- Slovic, P. (1987). Perception of risk. *Science*, 236(4799), 280–285.
- Slovic, P., Finucane, M. L., Peters, E., & MacGregor, D. G. (2002). The affect heuristic. In T. Gilovich, D. Griffin, & D. Kahneman (Eds.), *Heuristics and biases: The psychology of intuitive judgment* (pp. 397–420). Cambridge, England: Cambridge University Press.
- Small, D. A., & Lerner, J. S. (2005). *Emotional politics: Personal sadness and anger shape public welfare preferences*. Paper presented at the Society for Personality and Social Psychology, New Orleans, LA.
- Smith, C. A., & Ellsworth, P. C. (1985). Patterns of cognitive appraisal in emotion. *Journal of Personality and Social Psychology*, 48(4), 813–838.
- Smith, R. H., Turner, T. J., Garonzik, R., Leach, C. W., Urch, V., & Weston, C. (1996). Envy and schadenfreude. *Personality and Social Psychology Bulletin*, 22, 158–168.
- Solomon, R. C. (1990). *A passion for justice*. Reading, MA: Addison-Wesley Publishing Company.
- Spielberger, C. D. (1996). *Manual for the State-Trait Anger Expression Inventory (STAXI)*. Odessa, FL: Psychological Assessment Resources.
- Tamir, M., Mitchell, C., & Gross, J. J. (2008). Hedonic and instrumental motives in anger regulation. *Psychological Science*, 19, 324–328.
- Tavris, C. (1989). *Anger: The misunderstood emotion*. New York: Touchstone Books/Simon, & Schuster, Inc.
- Tiedens, L. Z. (2001a). Anger and advancement versus sadness and subjugation: The effect of negative emotion expressions on social status conferral. *Journal of Personality and Social Psychology*, 80(1), 86–94.
- Tiedens, L. Z. (2001b). The effect of anger on the hostile inferences of aggressive and nonaggressive people: Specific emotions, cognitive processing, and chronic accessibility. *Motivation and Emotion*, 25(3), 233–251.
- Tiedens, L. Z., & Linton, S. (2001). Judgment under emotional certainty and uncertainty: The effects of specific emotions on information processing. *Journal of Personality and Social Psychology*, 81, 973–988.
- Tooby, J., & Cosmides, L. (1990). The past explains the present: Emotional adaptations and the structure of ancestral environments. *Ethology and Sociobiology*, 11(4-5), 375–424.
- Tripp, T. M., & Bies, R. J. (1997). What's good about revenge? The avenger's perspective. In R. J. Lewicki, & R. J. Bies (Eds.), *Research on negotiation in organizations* (Vol. 6, pp. 145–160). New York: JAI Press.
- Tversky, A., & Kahneman, D. (1981). The framing of decisions and the psychology of choice. *Science*, 211(4481), 453–458.
- Weiner, B. (1980). A cognitive (attribution)-emotion-action model of motivated behavior: An analysis of judgments of help-giving. *Journal of Personality and Social Psychology*, 39(2), 186–200.
- Weiner, B. (1986). Attribution, emotion, and action. In R. M. Sorrentino, & E. T. Higgins (Eds.), *Handbook of motivation and cognition: Foundations of social behavior* (pp. 281–312). New York: Guilford Press.
- Williams, J. E., Patson, C. C., Siegler, I. C., Eigenbrodt, M. L., Nieto, F. J., & Tyroler, H. A. (2000). Anger proneness predicts coronary heart disease risk. *Circulation*, 101, 2034–2039.
- Wright, W. F., & Bower, G. H. (1992). Mood effects on subjective probability assessment. *Organizational Behavior and Human Decision Processes*, 52(2), 276–291.
- Young, M. J., & Tiedens, L. Z. Mad enough to see the other side: The effect of anger on hypothesis disconfirmation. Working Paper.

Biographical Author Sketch

Paul M. Litvak: Paul Litvak is a doctoral student in Behavioral Decision Research and Psychology at Carnegie Mellon University and is a research associate at the Harvard Decision Science Laboratory at Harvard University.

Jennifer S. Lerner: Jennifer Lerner is Professor of Public Policy and Management at the Harvard Kennedy School of Government. She also holds a faculty position in the Harvard Department of Psychology. Lerner is Founder and Director of the Harvard Decision Science Laboratory. The laboratory draws on psychology, economics, and neuroscience to study emotional influences on human judgment and decision-making.

Larissa Z. Tiedens: Larissa Tiedens is Professor of Organizational Behavior at the Stanford Graduate School of Business. Her research explores the role of emotions in social judgment and cognitive processing as well as the psychological dynamics of social hierarchies.

Katherine Shonk: Katherine Shonk is the editor of the *Negotiation* newsletter, published by the Program on Negotiation at Harvard Law School, and a research associate at Harvard Business School and Harvard's John F. Kennedy School of Government.

Chapter 18

State and Trait Anger, Fear, and Social Information Processing

David Schultz, Angela Grodack, and Carroll E. Izard

Abstract Emotions include a configuration of facial muscles, an arousal of physiological functions, and a facilitation of particular attention and information processing patterns (Izard, 1991). This chapter focuses on how anger influences attention and information processing. We will see that the most reliable effects of anger's influence on attention and information processing occur in anger-, hostile-, or aggression-prone individuals who are in an angry state. This trait–state interaction likely occurs because anger arousal in some ways limits processing of information, leading particular individuals to utilize processing lessons learned from past experiences of anger and threat. We discuss how and why different individuals may have different processing tendencies, especially related to the detection of anger and threat. We then review how and why other individuals might, as a product of their experiences of anger and threat, develop different anger- and threat-related processing schemas.

Several years ago the first author sat in a therapy office with a single mother and her 15-year-old son. The mother reported an incident in which, despite her request for him to do so, her son failed to clean up a room in their house. She reported feeling disrespected by this and for a couple of minutes listed other examples of ways in which her son disappointed or disrespected her. At one point I looked over at the son and noticed a slight grimace on his face. When he saw me look at him, he spoke up, “Mom, when you go on like this, I tune you out!” Despite her initial surprise at this statement, the son's ability to speak up for the first time about her mother's monologues eventually led, in part, to significant and positive change for the two of them. They became able to recognize and practice their conversation dynamics – including using a 1-min egg timer for mother and her monologues – that led to meaningful change in their interactions and relationship.

This example likely shows anger in action, both in an adaptive and in a less adaptive manner. At least in part, her son's annoyance at mother's self-righteous monologues seems to have motivated him to speak up. The anger mother felt because of her son's disrespect, however, seemed to motivate her not only to search her memory banks for supporting evidence for his disrespect but also to list the results to him at length.

Emotions are our brain's and body's “best guesses” as to how we should respond when certain categories of events occur, such as goal blockage or object loss (Tooby & Cosmides, 1990). These “best guesses” may include the configuration of facial muscles, arousal of physiological functions, and facilitation of particular attention and information processing patterns (Izard, 1991). The present chapter focuses on the last of these functions. In particular, we review evidence for how

D. Schultz (✉)

Department of Psychology, University of Maryland – Baltimore County, Baltimore, MD, 21250, USA
e-mail: dschultz@umbc.edu

anger relates to attention and information processing. We first address how anger arousal influences attention and information processing. We will see that the most reliable effects of anger's influence on attention and information processing occur in anger-, hostile-, or aggression-prone individuals who are in an angry state. This trait-state interaction likely occurs because anger arousal in some ways limits processing of information, leading particular individuals to utilize processing lessons learned from past experiences of anger and threat. To the extent that individuals have developed different processing tendencies because of different experiences of and exposure to anger and threat, we can expect them to display somewhat different processing tendencies when aroused by anger. We discuss how and why different individuals may have different processing tendencies, especially related to the detection of anger and threat. We begin with how anger-prone individuals tend to process social information. We then review how and why other individuals might, as a product of their experiences of anger and threat, develop different anger- and threat-related processing schemas.

18.1 Adaptive Function of Anger

The basic emotion of anger typically motivates the removal of obstacles to goals (Izard, 1991). Anger arousal occurs most consistently when one perceives another person or an object or event as causing the obstacle without justification (Smith & Ellsworth, 1985) and perceives an opportunity to change the anger-eliciting context (Harmon-Jones & Allen, 1998). The neural processes underlying a limited set of about six basic emotions (Ekman & Davidson, 1994; Izard, 1977; Panksep, 2000), including anger, are innate and universal (for an alternative, constructivist view, see Barrett, 2005). Basic anger, like other basic negative emotions, typically consists of a relatively brief emotion activation-feeling/motivational-action episode.

Beginning in the first year of life, as the infant develops higher-order cognition, 'pure' basic emotion responses appear less frequently (Izard, *in press*). Rather, higher-order cognition and perceptual processes (e.g., appraisals, expectations of responses) interact with emotion functions (i.e., expressive and feeling/motivational properties) to activate and regulate arousal and expression. Differential emotions theory (DET; Izard, *in press*) calls these interactions "emotion schemas."

Emotion schemas change an individual's emotion functioning. A limited number of perceptual stimuli, such as arm restraint, will activate basic anger. Based on individual, social, and cultural differences in anger schemas, however, many different stimuli may come to activate angry feelings (Izard, *in press*). Additionally, whereas a basic anger episode typically is relatively brief in duration, an anger schema may operate continually for indefinite periods of time, such as in the case of toddler temper tantrums (Potegal, Kosorok, & Davidson, 2003; see Chapter 22 for a review of the time course and dynamics of anger). Additionally, frequently occurring emotion schemas may stabilize to form traits of personality, such as positive emotionality/extraversion, fearfulness/inhibition (Clark, Watson, & Mineka, 1994; Rothbart & Bates, 1998; Goldsmith, Lemery, & Aksan, 2000), and the anger-hostility-aggression (AHA) syndrome discussed in Chapter 23.

Both basic anger and anger schemas serve the goal of obstacle removal. An angry feeling signals to an individual to search for and change the anger-eliciting context. An angry face typically serves as an alerting sign to others. An anger-related behavior usually takes the form of self-assertion, ranging from statements of appropriate self-assertion and defense of one's self to harmful aggressive actions. As we discuss anger's influence on attention and information processing, we should similarly expect its effects to serve obstacle removal.

18.2 Angry States and Attention and Information Processing

Empirical interest in how angry states influence attention and information processing is relatively recent. Most individuals who have investigated emotion influences on cognition have defined emotion by dimensions (i.e., by positive vs. negative valence) rather than discrete emotions such as anger, happiness, sadness, fear (e.g., Isen, 1990). A large body of research documents that arousal of negative emotions will influence interpretations of social stimuli (for a review, see Chapter 16 Rusting, 1998). Does the discrete emotion of anger influence attention and information processing? Yes, but research suggests that it does so much more strongly and consistently for some people than others. The following sections review these findings.

A few studies have examined the effects of anger arousal on attention. In an early study of anger perception, anger induced through role play tended to bias participants toward perceiving anger expressions when exposed stereoscopically to both anger and joy expressions (Izard, Wehmer, Livsey, & Jennings, 1965). More recently, Eckhardt and colleagues have conducted several studies examining anger's effects on attention. In one study anger arousal led to greater allocation of processing resources to anger-related words as measured by Stroop interference (Cohen, Eckhardt, & Schagat, 1998) in all subjects, but in another study it did not affect low-trait anger subjects (Eckhardt & Cohen, 1997). Individuals high in trait anger showed consistent effects in both studies, however. Following insult, these individuals dedicated greater early processing resources to anger-related words (Cohen et al., 1998; Eckhardt & Cohen, 1997).

Perhaps the most consistently demonstrated influence of anger to date involves interpretation of stimuli. Anger arousal seems to motivate interpretations of anger and hostility in others and heighten the significance an individual ascribes to an event. Adults who were angry or exposed to anger more likely perceived others' emotions and facial expressions as angry or negative (Schiffenbauer, 1974, cf. Carlson, Fellman, & Masters, 1983). Relatedly, angry and agitated individuals tend to search for causality and blameworthiness and attribute more negative intent to others' behaviors (Eckhardt & Jamison, 2002; Keltner, Ellsworth, & Edwards, 1993; Lerner, Goldberg, & Tetlock, 1998; Mayer & Hanson, 1995). And, anger induction can lead to irrational and otherwise distorted thoughts, such as catastrophizing, overgeneralization, and dichotomous thinking (Eckhardt, Barbour, & Davison, 1998; Eckhardt & Jamison, 2002; Zillman, 1994).

As with attention processes, anger's effects on interpretation are much more pronounced and consistently seen in certain individuals. Many studies find that angry states only influence interpretations for individuals high in trait anger, hostility, and/or aggression, which together constitute the anger-hostility-aggression (AHA) syndrome (Chapter 23). For example, following frustration because of loss at a computer game, nonaggressive and moderately aggressive boys did not attribute more hostile intent to protagonists (Orobio de Castro, Slot, Bosch, Koops, & Veerman, 2003). Anger only significantly influenced the attributions of highly aggressive boys. Similar results have been found with aggressive and nonaggressive college students (Tiedens, 2001). Other studies find that anger had a much more pronounced effect on college students high in trait anger than those low in trait anger (Guyl & Maddon, 2004) and on men with a history of marital violence than men with no such history (Eckhardt et al., 1998; Eckhardt & Jamison, 2002).

As described in detail elsewhere in this handbook, anger also seems to influence the decision making process (Chapter 17: "Fuel in the fire: How Anger Impacts Judgement and Decision Making"). In particular, anger motivates less attention to risk for negative outcomes and, compared to fear, more optimistic expectations of possible outcomes (Keltner et al., 1993; Lerner & Keltner, 2000). Relatedly, compared to a more neutral mood state, anger seems to motivate children to focus their goals on the re-acquisition of lost objects more than on interpersonal goals, such as whether or not peers like the child (Lemerise, Harper, Caverly, & Hobgood, 1998).

How might we explain these effects of anger on attention and information processing? Early theories suggested that discrete emotion experiences motivate processing biases in favor of the same emotion (Bower, 1981; Tomkins & McCarter, 1964). Similarly, others stated that anger “primes” harm-related emotion schemas such that individuals will attend to and interpret ambiguous information as angry, harmful, and threatening (Keltner et al., 1993).

More recent accounts have emphasized the functional nature of emotion experiences. As stated previously, anger largely functions to motivate obstacle removal. Angry states should therefore motivate attention and information processing patterns that serve this function. One way by which, many theorists suggest, anger generally accomplishes this goal is by its influence on the cognitive “space” involved in processing (Bodenhausen, 1993; Damasio, 1994; Lerner, Goldberg, & Tetlock, 1998; Oatley & Jenkins, 1996; Tiedens, 2001; Zillman, 1994). To facilitate a rapid response to goal obstruction and threat, anger narrows the cognitive space an individual searches to perceive and interpret stimuli and formulate cognitive representations of behavioral responses. This theoretical emphasis may help explain the stronger effects seen for anger arousal in high-trait anger individuals than in low-trait anger individuals. Because of the salience of emotion schemas related to the perception of anger and threat, when in an angry state individuals high in trait anger utilize these anger- and threat-related schemas. Individuals without as fully developed or salient processing schemas toward anger and threat will not as frequently utilize these schemas in an angry state.

What types of schemas do individuals high in trait anger have? We turn to this question now.

18.3 Trait Anger and Attention and Information Processing

Some individuals anger more frequently and intensely than others. Stable individual differences in anger arousal exist from the first year of life (Denham, Lehman, Moser, & Reeves, 1995; Izard, Libero, Putnam, & Haynes, 1993; Lemery, Goldsmith, Klinnert, & Mrazek, 1999; Sullivan, Lewis, & Alessandri, 1992). These individual differences seem to arise both from genetic and environmental influence. Twin studies suggest genetic differences make a moderate contribution to trait anger (for a review, see Chapter 3). And, as reviewed in other chapters in this handbook (Chapter 11), many individuals are socialized to express anger more frequently than others.

Many depressed individuals hold a “depressogenic” cognitive style (Mezulis, Hyde, & Abramson, 2006). In response to negative events, depressed individuals tend to attribute them to internal, stable, and pervasive aspects of their personalities and competencies. Do individuals high in trait anger similarly have characteristic processing styles? We believe the answer is “yes,” but we have a very incomplete picture at this point of what it entails. Researchers have examined trait anger much less frequently than trait anxiety or sadness (Parrott, Zeichner, & Evces, 2005). Many studies with adults have examined “hostility,” a cognitive style in which individuals dislike and mistrust others and attribute selfish and harmful intentions to their actions (Miller, Smith, Turner, Guijarro, & Hallet, 1996). Although trait anger correlates with hostility (Liehr, Meininger, Mueller, Chan, Fraizere, & Reyes, 2000; Smith, 1992; Woodall & Matthews, 1989), many theorists have emphasized the conceptual and empirical distinctness of these constructs (e.g., Gallo & Smith, 1997). In this section, we review evidence specifically for trait anger and its relation to attention and information processing tendencies.

Studies have documented that individuals high in trait anger exhibit early (i.e., attentional) information processing biases. In particular, they tend to orient to angry faces and anger-oriented information. On Stroop tasks, adults high in trait anger show more interference when presented with angry faces staring at them, suggesting increased orientation to these faces (Putnam, Hermans, &

van Honk, 2004; van Honk et al., 2003; van Honk et al., 2001; van Honk et al., 1998; van Honk et al., 1999). Similar results have also been found with anger-related words (Parrott et al., 2005; van Honk et al., 2001).

Some evidence suggests that individuals high in trait anger also exhibit interpretational biases. For example, more anger-prone children exhibit less accurate recognition of facial expressions (Arsenio, Cooperman, & Lover, 2000; Matsumoto, LeRoux, & Wilson-Cohn, 2000). This inaccuracy may very well reflect a particular processing bias in anger-prone children. We presented young children with ambiguous facial expressions and situational vignettes and asked them to label the protagonist's emotion states (Schultz, Izard, & Ackerman, 2000; Schultz, Izard, & Bear, 2004). First- and second-grade children higher on trait anger more frequently attributed anger to the facial expressions or vignettes. Similar results have been found with adult samples. In response to described situations, college students high in trait anger attributed more hostility to characters (Epps & Kendall, 1995) or more negativity to explanations of events (Wenzel & Lystad, 2005).

Why do individuals high in trait anger have these attention and information processing tendencies? Van Honk et al. (2003) suggest that in order to maintain dominance within their social environments angry individuals attempt to detect anger and threat quickly. When presented with angry faces gazing at them, individuals high in trait anger may more readily perceive this as challenging and allocate more processing resources to these faces. Other evidence suggests these interpretational biases may simply reflect tendencies for individuals with high-trait anger to expect that others will have feelings and motivations similar to theirs. Compared to individuals low in trait anger, individuals high in trait anger more quickly read sentences in which ambiguous provocation and subsequent angry reactions occur (Wingrove & Bond, 2005). Wingrove and Bond interpret this finding as suggesting that, following provocation, individuals high in trait anger more likely expected angry reactions and therefore more readily processed the sequences of action they read.

Might these associations more simply reflect the effects of anger arousal while completing experimental tasks, with high-trait anger individuals more likely to have experienced anger arousal during the cognitive tasks? The answer seems to be "no." Several of the aforementioned studies asked participants to rate their level of anger arousal during experimental tasks. Individuals high and low in trait anger did not differ in how angry they reported feeling during the tasks (e.g., Wingrove & Bond, 2005).

18.4 The Perception of Anger and Threat

We propose that angry states will have a relatively stronger effect on attention and information processing not just for individuals high in the AHA syndrome but for any individual for whom obstacle removal is relatively salient. Stated another way, anger arousal should influence attention and information processing more clearly in those individuals motivated to detect threat in their environments either more quickly or in a wider variety of situations. We propose this includes not only individuals high in trait anger who may be motivated to establish and maintain dominance in their social environments but also those with more fear-mediated experiences, such as those high in trait anxiety or those who have experienced stressful or abusive early environments. The detection of threat is a specific form of information processing that deserves special attention, and we turn to it now.

Many observers and researchers have noted that some children and adults more readily detect and infer anger and threat in their social environments (Dodge, 1980; Mogg, Philippot, & Bradley, 2004; Pollak, Cicchetti, Hornung, & Reed, 2000; Schultz et al., 2000). For example, imagine a fifth-grade boy who asks a classmate if he can borrow the classmate's pencil, and the classmate

says, “No.” In response to this reply, many boys will assume the classmate has a good reason not to lend the pencil or, at least, not make interpersonal inferences based on this interaction. Some other boys, however, will assume the classmate is angry at them or being mean toward them. Dodge and his colleagues have conducted extensive research on aggressive and peer-rejected children using scenarios similar to this one (for reviews, see Dodge, 2006; Orobio de Castro, Veerman, Koops, Bosch, & Monshouwer 2002). Compared to others, aggressive children, especially reactive or emotionally aggressive children, more frequently interpret ambiguous social provocation as “mean” and “hostile.” Challenging these types of attribution tendencies has played an important role in cognitive-behavioral treatments for adult anxiety, depression, and anger and become a focus of many violence prevention and intervention programs with children and adolescents (e.g., John Lochman’s Coping Power Program, The Committee for Children’s Second Step Violence Prevention Program).

Many other researchers have examined constructs similar to Dodge’s attribution of hostile intent. Downey and colleagues have examined “rejection sensitivity” in older children and adolescents, a construct focused on interpretations of social rejection (Downey, Mougios, Ayduk, London, & Shoda, 2004). Several researchers have used facial expression recognition procedures with younger children to examine perceptions of and sensitivity to anger in facial expressions (Barth & Bastiani, 1997; Cadesky, Mota, & Schachar, 2000; Schultz et al., 2000; Schultz et al., 2004; Pollak & Kistler, 2002). Still others have examined sensitivity to angry or threatening faces or words at a preconscious level, using either ERP amplitude (Pollak, Cichetti, & Klorman, 1998) or response latencies on Stroop tasks or dot probe tasks (Mogg et al., 2004).

We do not yet know if common underlying mechanisms account for all the effects these different methodologies produce. They share commonality in that they assess sensitivity to anger and threat. Yet, they differ on several dimensions: preconscious vs. conscious processing, judgments of personalized experiences vs. unknown others, and attributions of emotional states vs. intentions. These processing patterns have also been associated with individuals who behave very differently, such as those high in trait anger, as previously reviewed, but also those high in trait anxiety (Mogg et al., 2004). It remains unclear if a single theory might synthesize these results and account for these related phenomena. In the following sections we outline much of our understanding of the development of these phenomena. It should be noted at the outset, however, that a fuller understanding of these phenomena will require more empirical evidence, particularly from neuroscientific investigations of infant and toddler development.

18.4.1 Etiology and Development

Dodge (2006) recently suggested that, in response to social provocation, attributions of hostile intent (e.g., “he was being mean”) may reflect an earlier and more naturally developing form of information processing than more benign attributions or emotion schemas (e.g., “he isn’t angry at you,” “it was an accident”). He suggests that beginning at the age of 3 most children begin to learn to attribute benign intent to provocations. In this view, individual differences in attributions of hostile intent largely depend upon whether or not a child has learned to attribute benign intent instead of hostile intent.

We generally agree with this conceptualization but believe developmentally earlier processes explain individual differences more fully. To understand the development of individual differences in attributions of anger and threat, it is most critical to understand why some children are motivated to attribute causality and intention more than others. The same situation may provoke one child but

not another. Many infants and children who are bumped, for example, make little of it. A prerequisite for the attribution of anger and threat to others is motivation to search for causality and intention. We propose that heightened experiences of two emotions in the first several years of life – namely, anger and fear – likely lead some young children to learn to search for causality and intention in a wider variety of situations than most children.

Anger arousal tends to motivate a cognitive search for external causality and blame (Keltner et al., 1993). Much of the time, attributions of hostile intent to some extent reflect cognitive concomitants of anger. The child angers and therefore searches for cause and blameworthiness in others. Multiple studies find that either ratings of how angry provoked children feel (Camodeca & Goossens, 2005; Orobio de Castro, Merk, Koops, Veerman, & Bosch, 2005) or change in their autonomic arousal following provocation (Williams, Lochman, Phillips, & Barry, 2003) predict children's attributions of hostile intent in others. Children and adults with predispositions to anger or who have been socialized to anger in response to a greater variety of situational triggers will therefore more likely attribute anger and threat to others.

Relatedly, we have proposed that emotion processing patterns from the first several years of life likely lay a foundation upon which more specific and cognitively advanced forms of information processing, such as attributions of hostile intent, develop (Schultz, Abe, & Izard, 2005). Whereas a rudimentary ability to recognize some emotions in others appears by age 3–5 months (Izard, 1991; Montague & Walker-Andrews, 2002), understanding intentions apart from situational outcomes may not appear clearly until the fourth or fifth year of life (Flavell, Miller, & Miller, 1993). In the preschool years, when many advances in children's theory of mind occur, we expect a child's emotion processing tendencies to influence the development of individual differences in attributions of intent. For example, imagine a 4-year-old already sensitive to anger and threat. A foam soccer ball softly bounces off her back while she is walking around the playground. If cognitively able to infer intent, her tendency to perceive anger or threat in her environment will tend to motivate a search for cause and/or blame that may (and, at this age, will likely) end with an attribution of hostile intent. If placed in the same situation, another 4-year-old without as strong a sensitivity to anger and threat may not allocate processing resources to determine the source of this minor physical provocation.

Previous experiences of fear and threat may be as important as anger experiences, if not more so, to the development of easily aroused anger and threat processing patterns. For example, in our work children's trait anger predicted their attributions of anger to ambiguous facial expressions significantly but moderately (Schultz et al., 2004), but family stress predicted these processing biases more strongly (Schultz et al., 2000). The next sections review evidence for the role of fear in interpretational biases.

18.4.2 Fear and the Perception of Anger and Threat

Much empirical evidence demonstrates sensitivity among anxious individuals to the detection of anger and threat (Vasey & MacLeod, 2001; Williams, Mathews, & MacLeod, 1996; for a review, see Williams, Watts, MacLeod, & Matthews, 1997). Interestingly, several studies suggest anxious adults initially orient toward angry faces followed by an avoidance of the angry (and presumably threatening) face. For example, on dot probe tasks, anxious adults have exhibited increased attention to angry faces at 500 ms but not at longer intervals (Bradley, Mogg, White, Groom, & de Bono, 1999; Gotlib, Krasnoperova, Yue, & Joormann, 2004; Mogg et al., 2004). These

findings suggest that, among fearful and anxious individuals, processing biases occur at an attentional level and likely outside of reflective consciousness (Block, 2005). Anxiety-related biases are also found at an interpretational level and thus within reflective consciousness. For example, anxious adults rate ambiguous situations as more threatening (Butler & Mathews, 1983; MacLeod & Cohen, 1993) and explain ambiguous events more negatively (Wenzel & Lystad, 2005). Two studies have found that aggressive boys did not attribute hostile intent to provocateurs unless previously induced to feel anxiety (Dodge & Somberg, 1987; Williams et al., 2003). Moreover, compared to adults receiving a placebo, normal adults who took anti-anxiety medication attributed less anger and fear to facial expressions (Harmer, Shelley, Cowen, & Goodwin, 2004).

The previous results suggest commonality in early processing patterns between anger-prone and fear-prone individuals. This commonality likely helps explain a wealth of empirical evidence that suggests a close association between anger and fear (Berkowitz & Harmon-Jones, 2004; for a review, see Barlow, 2002). For example, ratings of trait anger and trait fear relate positively (Emmons & Diener, 1985), and we often experience anger and fear simultaneously or subsequent to one another (Diener & Iran-Nejad, 1986; Sherer & Tannenbaum, 1986). In children, for example, experiences of threat, such as parental conflict, often elicit a mixture of both anger and fear (Cummings, Iannotti, & Zahn-Waxler, 1985, 1989, 1991, 1993). And, interestingly, panic attacks in the abuser may sometimes motivate domestic violence (Mitchell & Gilchrist, 2006).

Growing evidence about the amygdala may help account for some of the conceptual and empirical association between anger and fear arousal. Some theorists suggest that the amygdala plays a particularly important role in the detection of threat (Adolphs & Tranel, 2004). The amygdala receives both direct and indirect projections from the sensory cortices (LeDoux, 2000). Because of these direct projections, the amygdala facilitates rapid, automatic detection of threat. Additionally, however, the amygdala itself projects not only to higher cortical regions but also back to the sensory cortices (Amaral, Price, Pitkinson, & Carmichael, 1992; Amaral & Price, 1984). Because of this, Adolphs (2003) suggests that the amygdala is “anatomically positioned” to have great influence on both how we interpret what we perceive and what we attend to in the first place. Several interesting studies support the potential role for the amygdala in anger and threat detection. For example, adults with an intact amygdala rated facial expressions as less trustworthy and approachable than those with bilateral amygdala damage (Adolphs, Tranel, & Damasio, 1998). Relatedly, the amygdala is activated when making judgments of untrustworthiness (Winston, Strange, O’Doherty, & Dolan, 2002). Some evidence demonstrates that amygdala activation occurs outside of reflective awareness. For example, amygdala activation occurred even when angry facial expressions were only presented for milliseconds and then quickly masked with a neutral face (Rauch, Whalen, Shin, et al., 2000). Although these results are suggestive of the role of the amygdala in anger and fear, the amygdala is also activated when processing happy and sad faces (Yang et al., 2002). It remains unclear if the amygdala’s role is primarily specific to anger and threat processing or involved in more general processes, such as detecting distress (Blair, Morris, & Frith, 1999) or withdrawal-related information (Anderson, Spencer, Fulbright, & Phelps, 2000).

The amygdala is likely the anatomical substrate of threat processing described in some cognitive models. For example, in Williams et al.’s (1996) revision of Cohen, Dunbar, and McClelland (1990) parallel distributed processing model, they propose that for each individual, certain stimuli are “tagged” as threatening. Perception of a tagged stimulus activates a resource allocation mechanism (RAM) which directs attention to the stimuli and certain aspects of it. The amygdala seems central to this process, and, as stated previously, seems related to “tagging” stimuli outside of reflective consciousness.

18.4.3 Socialization of Threat

Tagging seems to occur primarily as a result of experiences of fear and anger. When a threatening event occurs, it's the fear and/or anger associated with it – an aversive state – that motivates “tagging” stimuli for future early detection. By detecting tagged stimuli early on within interactions, the individual may potentially avoid fear- and/or anger-arousing events (see Chapter 15). Both theory and work with physically abused preschool children support these concepts (Pollak, Vardi, Bechner, & Curtin, 2005). Pollak suggests that experiences of threat and young children's limited capacity to attend to multiple stimuli partner to shape processing biases in these children. In a series of studies, Pollak and his colleagues have demonstrated that physically abused preschool children have both attention and interpretational biases toward angry faces (Pollak et al., 1998; Pollak & Kistler, 2002; Pollak & Sinha, 2002; Pollak & Tolley-Schell, 2003; Pollak et al., 2003; Pollak et al., 2000). And, they have demonstrated this bias with multiple methodologies. Physically abused children show greater p300 amplitude when searching for angry faces (suggesting a heightened salience of angry faces to them; Pollak & Tolley-Schell, 2003), take longer to disengage from the search for an angry face (Pollak & Tolley-Schell, 2003), require less visual information to detect an angry face (Pollak & Sinha, 2002), and, in response to faces with multiple emotion cues, attribute anger to these faces more often than comparison children (Pollak & Kistler, 2002). Interestingly, in this last study, results suggested that non-abused children biased their interpretations away from anger. In response to morphed facial expressions ranging from, for example, a prototypic fear expression to a prototypic anger expression, physically abused children's emotion attributions seemed closer to reality. Whereas non-abused children on average did not attribute “anger” to the perceived facial expression until about 70% of the facial cues indicated anger (vs. 30% indicating fear), physically abused children attributed anger closer to the 50/50 split of anger/fear cues, on average attributing anger with about 40% anger cues (Pollak & Kistler, 2002).

Findings from other studies converge with those of Pollak and colleagues. In our own work with a community sample of preschool children, those living with lower-income families most characterized by instability and stress – such as frequent residence changes, job changes, relationship changes, caregiver depression – also tended to interpret ambiguous faces and characters in described situations as feeling angry (Schultz et al., 2000). Using the attribution of intent methodology, children whose caregivers either physically abused them or otherwise disciplined them harshly (e.g., slapping, spanking) attributed more hostility to hypothetical others than did their peers (Dodge, Pettit, Bates, & Valente, 1995; Weiss, Dodge, Bates, & Pettit, 1992; cf. Heidgerken, Hughes, Cavell, & Willson, 2004). Other disparate studies provide indirect support by finding that (1) children from disrupted (vs. two-parent) homes more frequently attributed “anger” to facial expressions (Reichenbach & Masters, 1983), (2) insecure infant attachment predicted more frequent attributions of “anger” and “insensitivity” to mothers in middle childhood (Ziv, Oppenheim, & Sagi-Schwartz, 2004), and (3) preschool children exposed to higher levels of community violence generally recognized emotions less accurately (Farver, Xu, Eppe, Fernandez, & Schwartz, 2005).

18.4.4 Other Correlates of the Processing of Anger and Threat

Thus far we have emphasized the role of anger and fear arousal and a history of anger and fear experiences in tendencies to process anger and threat. Clearly other factors also play a role and will influence the extent to which anger arousal influences attention and information processing. First, positive emotional experiences likely influence processing. Seventh-grade children induced to

feel a positive mood through music attributed less hostility in response to characters in hypothetical situations than those in other moods (Bryan, Sullivan-Burnstein, & Mathur, 1998). When in an angry state, children who are generally happy may therefore attribute less anger or threat to others.

It remains unclear if, and how strongly a role, simple exposure to frequent anger and/or threat plays in processing biases toward anger and threat. We agree with others (e.g., Dodge, 2006) that the personal experience of anger and fear likely influences the development of these processing biases more strongly than modeling does. Maternal attributions of hostile intent predict child attributions of hostile intent (MacBrayer, Milich, & Hundley, 2003; MacKinnon-Lewis, Volling, Lamb, Dechman, Rabiner, & Curtner, 1994), particularly girls' (Bickett, Milich, & Brown, 1996; MacBrayer, Milich, & Hundley, 2003; MacKinnon-Lewis, Lamb, Arbuckle, Baradaran, & Volling, 1992). However, in these studies we do not know the process by which these children developed tendencies to attribute hostile intent. Modeling, personal experiences, both, or neither may help account for children's attribution tendencies.

18.5 Conclusion

Empirical findings to date suggest the relationship between anger and attention and information processing is not straightforward. In general, anger arousal should motivate attention and information processing that facilitates obstacle removal. Available evidence to date is limited but generally supports this conception. Anger seems to motivate the allocation of attention resources to angry and threatening cues, bias interpretation of stimuli as threatening and of great significance, and influence decision making toward action (e.g., expecting positive outcomes to occur from behavioral responses). The empirical findings for anger arousal, however, are more consistent for individuals high in the AHA syndrome than others. These findings suggest that anger arousal does not motivate specific thoughts so much as a style of utilizing existing schemas (e.g., pre-emptive processing). To the extent that individuals hold different processing schemas, anger arousal will influence attention and information processing differently. In addition to individuals high in the AHA syndrome, we suggest that differential effects of anger arousal might also occur for other individuals who hold easily aroused processing schemas toward anger and threat detection, such as abused individuals. It remains for future studies to examine this suggestion and, more generally, to develop our limited database of knowledge on how, why, and for whom anger arousal influences attention and information processing.

References

- Adolphs, R. (2003). Cognitive neuroscience of human social behaviour. *Nature*, 4, 165–178.
- Adolphs, R., & Tranel, D. (2004). Impaired judgments of sadness but not happiness following bilateral amygdale damage. *Journal of Cognitive Neuroscience*, 16, 453–462.
- Adolphs, R., Tranel, D., & Damasio, A. R. (1998). The human amygdale in social judgment. *Nature*, 393, 470–474.
- Amaral, D. G., & Price, J. L. (1984). *Journal of Computational Neurology*, 230, 465–496.
- Amaral, D. G., Price, J. L., Pitkinson, A., & Carmichael, S. T. (1992). Anatomical organization of the primate amygdaloid complex. In J. P. Aggleton (Ed.), *The amygdala: Neurobiological aspects of emotion, memory, and mental dysfunction* (pp. 1–66). New York: Wiley.
- Anderson, A. K., Spencer, D. D., Fulbright, R. K., & Phelps, E. A. (2000). Contribution of the anteromedial temporal lobes to the evaluation of facial emotion. *Neuropsychology*, 14, 526–536.
- Arsenio, W. F., Cooperman, S., & Lover, A. (2000). Affective predictors of preschoolers' aggression and peer acceptance: Direct and indirect effects. *Developmental Psychology*, 36, 438–448.

- Barlow, D. H. (2002). *Anxiety and its disorders: The nature and treatment of anxiety and panic*. 2nd ed. New York, NY: Guilford Press.
- Barrett, L. F. (2006). Are emotions natural kinds? *Perspectives on Psychological Science*, 1, 28–58.
- Barth, J. M., & Bastiani, A. (1997). A longitudinal study of emotion recognition and preschool children's social behavior. *Merrill-Palmer Quarterly*, 43, 107–128.
- Berkowitz, L., & Harmon-Jones, E. (2004). More thoughts about anger determinants. *Emotion*, 4, 151–155.
- Bickett, L. R., Milich, R., & Brown, R. T. (1996). Attributional styles of aggressive boys and their mothers. *Journal of Abnormal Psychology*, 105, 457–472.
- Blair, R. J. R., Morris, J. S., & Frith, C. C. (1999). Dissociable neural responses to facial expressions of sadness and anger. *Brain: A Journal of Neurology*, 122(5), 883–893.
- Block, N. (2005). Two neural correlates of consciousness. *Trends in Cognitive Sciences*, 9, 46–52.
- Bodenhausen, G. V. (1993). Emotions, arousal, and stereotypic judgments: A heuristic model of affect and stereotyping. In Mackie, D. M., & Hamilton, D. L. (Eds.), *Affect, cognition, and stereotyping: Interactive processes in group perception* (pp. 13–37). San Diego, CA: Academic Press.
- Bower, G. H. (1981). Mood and memory. *American Psychologist*, 36, 129–148.
- Bradley, B. P., Mogg, K., White, J., Groom, C., & de Bono, J. (1999). Attention bias for emotional faces in generalized anxiety disorder. *British Journal of Clinical Psychology*, 38, 267–278.
- Bryan, T., Sullivan-Burstein, K., & Mathur, S. (1998). The influence of affect on social-information processing. *Journal of Learning Disabilities*, 31, 418–426.
- Butler, G., & Mathews, A. (1983). Cognitive processes in anxiety. *Advances in Behaviour Research, & Therapy*, 5, 51–62.
- Cadesky, E. B., Mota, V. L., & Schachar, R. J. (2000). Beyond words: How do problem children with ADHD and/or conduct problems process nonverbal information about affect? *Journal of the American Academy of Child, & Adolescent Psychiatry*, 39, 1160–1167.
- Camodeca, M., & Goossens, F. A. (2005). Aggression, social cognitions, anger and sadness in bullies and victims. *Journal of Child Psychology and Psychiatry*, 46, 186–197.
- Carlson, C. R., Felleman, E. S., & Masters, J. C. (1983). Influence of children's emotion states on the recognition of emotion in peers and social motives to change another's emotional state. *Motivation and Emotion*, 7, 61–79.
- Clark, L. A., Watson, D., & Mineka, S. (1994). Temperament, personality, and the mood and anxiety disorders. *Journal of Abnormal Psychology*, 103(1), 103–116.
- Cohen, J. D., Dunbar, K., & McClelland, J. L. (1990). On the control of automatic processes: A parallel distributed processing account of the Stroop effect. *Psychological Review*, 97, 332–361.
- Cohen, D. J., Eckhardt, C. I., & Schagat, K. D. (1998). Attention allocation and habituation to anger-related stimuli during a visual search task. *Aggressive Behavior*, 24, 399–409.
- Cummings, E. M., Ballard, M., El-Sheikh, M., & Lake, M. (1991). Resolution and children's responses to interadult anger. *Developmental Psychology*, 27, 462–470.
- Cummings, E. M., Iannotti, R. J., & Zahn-Waxler, C. (1985). Influence of conflict between adults on the emotions and aggression of young children. *Developmental Psychology*, 21, 495–507.
- Cummings, E. M., Simpson, K. S., & Wilson, A. (1993). Children's responses to interadult anger as a function of information about resolution. *Developmental Psychology*, 29, 978–985.
- Cummings, E. M., Vogel, D., Cummings, J. S., & El-Shiekh, M. (1989). Children's responses to different forms of expression of anger between adults. *Child Development*, 60, 1392–1404.
- Damasio, A. (1994). *Descartes' error: Emotions, reason, and the human brain*. New York: Avon Books.
- Denham, S. A., Lehman, E. B., Moser, M. H., & Reeves, S. L. (1995). Continuity and change in emotional components of infant temperament. *Child Study Journal*, 25, 289–308.
- Diener, E., & Iran-Nejad, A. (1986). The relationship in experience between various types of affect. *Journal of Personality and Social Psychology*, 50, 1031–1038.
- Dodge, K. A. (1980). Social cognition and children's aggressive behavior. *Child Development*, 51, 162–170.
- Dodge, K. A. (2006). Translational science in action: Hostile attributional style and the development of aggressive behavior problems. *Development and Psychopathology*, 18, 791–814.
- Dodge, K. A., Pettit, G. S., Bates, J. E., & Valente, E. (1995). Social information-processing patterns partially mediate the effect of early physical abuse on later conduct problems. *Journal of Abnormal Psychology*, 104, 632–643.
- Dodge, K. A., & Somberg, D. R. (1987). Hostile attributional biases among aggressive boys are exacerbated under conditions of threat to the self. *Child Development*, 58, 213–224.
- Downey, G., Mougios, V., Ayduk, O., London, B. E., & Shoda, Y. (2004). Rejection sensitivity and the defensive motivational system: Insights from the startle response to rejection cues. *Psychological Science*, 15, 668–673.

- Eckhardt, C. I., Barbour, K. A., & Davison, G. C. (1998). Articulated thoughts of martially violent and nonviolent men during anger arousal. *Journal of Consulting and Clinical Psychology, 66*, 259–269.
- Eckhardt, C. I., & Cohen, D. J. (1997). Attention to anger-relevant and irrelevant stimuli following naturalistic insult. *Personality and Individual Differences, 23*, 619–629.
- Eckhardt, C., & Jamison, T. R. (2002). Articulated thoughts of male dating violence perpetrators during anger arousal. *Cognitive Therapy and Research, 26*, 289–308.
- Ekman, P., & Davidson, R. J. (1994). *The nature of emotion: Fundamental questions*. New York, NY: Oxford University Press.
- Emmons, R. A., & Diener, E. (1985). Personality correlates of subjective well-being. *Personality and Social Psychology Bulletin, 11*, 89–97.
- Epps, J., & Kendall, P. C. (1995). Hostile attributional bias in adults. *Cognitive Therapy and Research, 19*, 159–178.
- Farver, J. M., Xu, Y., Eppe, S., Fernandez, A., & Schwartz, D. (2005). Community violence, family conflict, and preschoolers' socioemotional functioning. *Developmental Psychology, 41*, 160–170.
- Flavell, J. H., Miller, P. H., & Miller, S. A. (1993). *Cognitive development*. 3rd ed. Upper Saddle River, NJ: Prentice Hall.
- Gallo, L. C., & Smith, T. W. (1997). Construct validation of health-relevant personality traits: Interpersonal circumplex and five-factor analyses of the Aggression Questionnaire. *International Journal of Behavioral Medicine, 5*, 129–147.
- Goldsmith, H. H., Lemery, K. S., & Aksan, N. (2000). Temperamental substrates of personality. In V. J. Molfese, & D. L. Molfese (Eds.), *Temperament and personality development across the life span* (pp. 1–32). Mahwah, NJ: Lawrence Erlbaum.
- Gotlib, I. H., Krasnoperova, E., Yue, D. N., & Joormann, J. (2004). Attention biases for negative interpersonal stimuli in clinical depression. *Journal of Abnormal Psychology, 113*, 127–135.
- Guyll, M., & Madon, S. J. (2004). Effects of trait hostility and self-relevance on social information processing. *Basic and Applied Social Psychology, 26*, 263–276.
- Harmer, C. J., Shelley, N. C., Cowen, P. J., & Goodwin, G. M. (2004). Increased positive versus negative affective perception and memory in healthy volunteers following selective serotonin and norepinephrine reuptake inhibition. *American Journal of Psychiatry, 161*, 1256–1263.
- Harmon-Jones, E., & Allen, J. J. (1998). Anger and frontal brain activity: EEG asymmetry consistent with approach motivation despite negative affect valence. *Journal of Personality and Social Psychology, 74*, 1310–1316.
- Heidergerken, A. D., Hughes, J. N., Cavell, T. A., & Willson, V. L. (2004). Direct and indirect effects of parenting and children's goals on child aggression. *Journal of Clinical Child and Adolescent Psychology, 33*, 684–693.
- Isen, A. M. (1990). The influence of positive and negative affect on cognitive organization: Some implications for development. In Stein, N. L., Leventhal, B., & Trabasso, T. (Eds.), *Psychological and biological approaches to emotion* (pp. 75–94). Hillsdale, NJ: Lawrence Erlbaum Associates.
- Izard, C. E. (1977). *Human emotions*. New York: Plenum Press.
- Izard, C. E. (1991). *The psychology of emotions*. New York: Plenum Press.
- Izard, C. E., Libero, D. Z., Putnam, P., & Haynes, O. M. (1993). Stability of emotion experiences and their relations to traits of personality. *Journal of Personality and Social Psychology, 64*, 847–860.
- Izard, C. E., Wehmer, G. M., Livsey, W., & Jennings, J. R. (1965). Affect, awareness, and performance. In S. S. Tomkins, & C. E. Izard (Eds.), *Affect, cognition, and personality* (pp. 2–41). New York: Springer.
- Izard, C. E. (2007). Basic emotions, natural kinds, emotion schemas, and a new paradigm, *Perspectives on Psychological Science, 2*, 260–280.
- Keltner, D., Ellsworth, P. C., & Edwards, K. (1993). Beyond simple pessimism: Effects of sadness and anger on social perception. *Journal of Personality and Social Psychology, 64*, 740–752.
- LeDoux, J. E. (2000). Emotion circuits in the brain. *Annual Review of Neuroscience, 23*, 155–184.
- Lemerise, E. A., Harper, B. D., Caverly, S., & Hobgood, C. (1998, March). Mood, social goals, and children's outcome expectations. Poster presented at Conference on Human Development, Mobile, AL.
- Lemery, K. S., Goldsmith, H. H., Klinnert, M. D., & Mrazek, D. A. (1999). Developmental models of infant and childhood temperament. *Developmental Psychology, 35*, 189–204.
- Lerner, J. S., Goldberg, J. H., & Tetlock, P. E. (1998). Sober second thought: The effects of accountability, anger, and authoritarianism on attributions of responsibility. *Personality and Social Psychology Bulletin, 24*, 563–574.
- Lerner, J. S., & Keltner, D. (2000). Beyond valence: Toward a model of emotion-specific influences on judgment and choice. *Cognition, & Emotion, 14*, 473–493.
- Liehr, P., Meininger, J. C., Mueller, W. H., Chan, W., Frazier, L., & Reyes, L. R. (2000). Psychometric testing of the adolescent version of the Cook-Medley Hostility Scale. *Issues in Comprehensive Pediatric Nursing, 23*, 103–116.

- MacBrayer, E. K., Milich, R., & Hundley, M. (2003). Attributional biases in aggressive children and their mothers. *Journal of Abnormal Psychology, 112*, 698–708.
- MacKinnon-Lewis, C., Lamb, M. E., Arbuckle, B., Baradaran, L. P., & Volling, B. L. (1992). The relationship between biased maternal and filial attributions and the aggressiveness of their interactions. *Development and Psychopathology, 4*, 403–415.
- MacKinnon-Lewis, C., Volling, B. L., Lamb, M. E., Dechman, K., Rabiner, D., & Curtner, M. E. (1994). A cross-contextual analysis of boys' social competence: From family to school. *Developmental Psychology, 30*, 325–333.
- MacLeod, C., & Cohen, I. L. (1993). Anxiety and the interpretation of ambiguity: A text comprehension study. *Journal of Abnormal Psychology, 102*, 238–247.
- Matsumoto, D., LeRoux, J., & Wilson-Cohn, C. (2000). A new test to measure emotion recognition ability: Matsumoto and Ekman's Japanese and Caucasian Brief Affect Recognition Test (JACBERT). *Journal of Nonverbal Behavior, 24*, 179–209.
- Mayer, J. D., & Hanson, E. (1995). Mood-congruent judgment over time. *Personality and Social Psychology Bulletin, 21*, 237–244.
- Mezulis, A. H., Hyde, J. H., & Abramson, L. Y. (2006). The developmental origins of cognitive vulnerability of depression: Temperament, parenting, and negative life events in childhood s contributors to a negative cognitive style. *Developmental Psychology, 42*, 1012–1025.
- Miller, T. Q., Smith, T. W., Turner, C. W., Guijarro, M. L., & Hallet, A. J. (1996). Meta-analytic review of research on hostility and physical health. *Psychological Bulletin, 119*, 322–348.
- Mitchell, I. J., & Gilchrist, E. (2006). Domestic violence and panic attacks – common neural mechanisms? *Legal and Criminological Psychology, 11*, 267–282.
- Mogg, K., Philippot, P., & Bradley, B. P. (2004). Selective attention to angry faces in clinical social phobia. *Journal of Abnormal Psychology, 113*, 160–165.
- Montague, D. P. F., & Walker-Andrews, A. S. (2002). Mothers, fathers, and infants: The role of person familiarity and parental involvement in infants' perception of emotion expressions. *Child Development, 73*, 1339–1352.
- Oatley, K., & Jenkins, J. M. (1996). *Understanding emotions*. Malden, MA: Blackwell Publishing.
- Orobio de Castro, B., Merk, W., Koops, W., Veerman, J. W., & Bosch, J. D. (2005). Emotions in social information processing and their relations with reactive and proactive aggression in referred aggressive boys. *Journal of Clinical Child and Adolescent Psychology, 34*, 105–116.
- Orobio de Castro, B., Slot, N. W., Bosch, J. D., Koops, E., & Veerman, J. W. (2003). Negative feelings exacerbate hostile attributions of intent in highly aggressive boys. *Journal of Clinical Child and Adolescent Psychology, 32*, 56–65.
- Orobio de Castro, B., Veerman, J. W., Koops, W., Bosch, J. D., & Monshouwer, H. J. (2002). Hostile attribution of intent and aggressive behavior: A meta-analysis. *Child Development, 73*, 916–934.
- Panksepp, J. (2000). Emotions as natural kinds within the mammalian brain. In M. Lewis, & J. M. Haviland-Jones (Eds.), *Handbook of emotions*. 2nd ed (pp. 137–156). New York: Guilford.
- Parrott, D. J., Zeichner, A., & Evces, M. (2005). Effect of trait anger on cognitive processing of emotional stimuli. *Journal of General Psychology, 132*, 67–80.
- Pollak, S. D., Cicchetti, D., Hornung, K., & Reed, A. (2000). Recognizing emotion in faces: Developmental effects of child abuse and neglect. *Developmental Psychology, 36*, 679–688.
- Pollak, S. D., Cicchetti, D., & Klorman, R. (1998). Stress, memory, and emotion: Developmental considerations from the study of child maltreatment. *Development and Psychopathology, 10*, 811–828.
- Pollak, S. D., & Kistler, D. J. (2002). Early experience is associated with the development of categorical representations for facial expressions of emotion. *Proceedings of the National Academy of Sciences, 99*, 9072–9076.
- Pollak, S. D., & Sinha, P. (2002). Effects of early experience on children's recognition of facial displays of emotion. *Developmental Psychology, 38*, 784–791.
- Pollak, S. D., & Tolley-Schell, S. A. (2003). Selective attention to facial emotion in physically abused children. *Journal of Abnormal Psychology, 112*, 323–338.
- Pollak, S. D., Vardi, S., Bechner, A. M., & Curtin, J. J. (2005). Physically abused children's regulation of attention in response to hostility. *Child Development, 76*, 968–977.
- Potegal, M., Kosorok, M. R., & Davidson, R. J. (2003). Temper tantrums in young children: 2. Tantrum duration and temporal organization. *Journal of Developmental, & Behavioral Pediatrics, 24*, 148–154.
- Putnam, P., Hermans, E., & van Honk, J. (2004). Emotional Stroop performance for masked angry faces: It's BAS, not BIS. *Emotion, 4*, 305–311.
- Rauch, S. L., Whalen, P. J., Shin, L. M., McInerney, S. C., Macklin, M. L., Lasko, N. B., et al. (2000). Exaggerated amygdale response to masked facial stimuli in posttraumatic stress disorder: A functional MRI study. *Biological Psychiatry, 47*, 769–776.

- Reichenbach, L., & Masters, J. C. (1983). Children's use of expressive and contextual cues in judgments of emotion. *Child Development*, 54, 993–1004.
- Rothbart, M. K., & Bates, J. E. (1998). Temperament. In W. Damon, & N. Eisenberg (Eds.), *Handbook of child psychology: Vol 3. Social, emotional, and personality development* (pp. 105–176). Hoboken, NJ: John Wiley, & Sons.
- Rusting, C. L. (1998). Personality, mood, and cognitive processing of emotional information: Three conceptual frameworks. *Psychological Bulletin*, 124, 165–196.
- Scherer, K. R., & Tannenbaum, P. H. (1986). Emotional experiences in everyday life: A survey approach. *Motivation and Emotion*, 10(4), 295–314.
- Schiffenbauer, A. (1974). Effect of observer's emotional state on judgments of the emotional state of others. *Journal of Personality and Social Psychology*, 30, 31–315.
- Schultz, D., Izard, C. E., & Abe, J. A. (2005). The emotion systems and the development of emotional intelligence. In *Emotional intelligence: An international handbook* (pp. 51–67). Ashland, OH: Hogrefe, & Huber Publishers.
- Schultz, D., Izard, C. E., & Ackerman, B. P. (2000). Children's anger attribution biases: Relations to family environment and social adjustment. *Social Development*, 9, 284–301.
- Schultz, D., Izard, C. E., & Bear, G. (2004). Children's emotion processing: Relations to emotionality and aggression. *Development, & Psychopathology*, 16(2), 371–387.
- Smith, T. W. (1992). Hostility and health: Current status of a psychosomatic hypothesis. *Health Psychology*, 11, 139–150.
- Smith, C. A., & Ellsworth, T. C. (1985). Patterns of cognitive appraisal in emotion. *Journal of Personality and Social Psychology*, 48, 813–838.
- Sullivan, M. W., Lewis, M., & Alessandri, S. M. (1992). Cross-age stability in emotional expressions during learning and extinction. *Developmental Psychology*, 28, 58–63.
- Tiedens, L. Z. (2001). The effect of anger on the hostile inferences of aggressive and nonaggressive people: Specific emotions, cognitive processing, and chronic accessibility. *Motivation and Emotion*, 25, 233–251.
- Tomkins, S. S., & McCarter, R. (1964). What and where are the primary affects? Some evidence for a theory. *Perceptual and Motor Skills*, 18, 119–158.
- Tooby, J., & Cosmides, L. (1990). The past explains the present: Emotional adaptations and the structure of ancestral environment. *Ethology and Sociobiology*, 11, 375–424.
- Van Honk, J., Kessels, R. P., Putnam, P., Jager, G., Koppeschaar, H. P., & Postma, A. (2003). Attentionally modulated effects of cortisol and mood on memory for emotional faces in healthy young males. *Psychoneuroendocrinology*, 28, 941–948.
- Van Honk, J., Tuiten, A., Verbaten, R., van den Hout, M., Koppeschaar, H., et al. (1999). Correlations among salivary testosterone, mood, and selective attention to threat in humans. *Hormones and Behavior*, 36, 17–24.
- Van Honk, J., Tuiten, A., van den Hout, M., Koppeschaar, H., Thijssen, J., et al. (1998). Baseline salivary cortisol levels and preconscious selective attention for threat: A pilot study. *Psychoneuroendocrinology*, 23, 741–747.
- Van Honk, J., Tuiten, A., van den Hout, M., Putnam, P., de Haan, E., & Stam, H. (2001). Selective attention to unmasked and masked threatening words: Relationships to trait anger and anxiety. *Personality and Individual Differences*, 30, 711–720.
- Vasey, M. W., & MacLeod, C. (2001). Information-processing factors in childhood anxiety: A review and developmental perspective. In *The developmental psychopathology of anxiety* (pp. 253–277). New York, NY: Oxford University Press.
- Weiss, B., Dodge, K. A., Bates, J. E., & Pettit, G. S. (1992). Some consequences of early harsh discipline: Child aggression and a maladaptive social information processing style. *Child Development*, 63, 1321–1335.
- Wenzel, A., & Lystad, C. (2005). Interpretation biases in angry and anxious individuals. *Behaviour Research and Therapy*, 43, 1045–1054.
- Williams, S. C., Lochman, J. E., Phillips, N. C., & Barry, T. D. (2003). Aggressive and nonaggressive boys' physiological and cognitive processes in response to peer provocations. *Journal of Clinical Child and Adolescent Psychology*, 32, 568–576.
- Williams, J. M., Mathews, A., & MacLeod, C. (1996). The emotional Stroop task and psychopathology. *Psychological Bulletin*, 120, 3–24.
- Williams, J. M., Watts, F. N., MacLeod, C., & Matthews, A. (1997). *Cognitive psychology and emotional disorders*. 2nd edition. Chichester, England: Wiley.
- Wingrove, J., & Bond, A. J. (2005). Correlation between trait hostility and faster reading times for sentences describing angry reactions to ambiguous situations. *Cognition, & Emotion*, 19, 463–472.
- Winston, J. S., Strange, B. A., O'Doherty, J., & Dolan, R. J. (2002). Automatic and intentional brain responses during evaluation of trustworthiness of faces. *Nature Neuroscience*, 5, 277–283.

- Woodall, K. L., & Matthews, K. A. (1989). Familial environment associated with Type A behaviors and psychophysiological responses to stress in children. *Health Psychology*, 8, 403–426.
- Yang, T. T., Menon, V., Eliez, S., Blasey, C., White, C. D., Reid, A. J., et al. (2002). Amygdalar activation associated with positive and negative facial expressions. *Neuroreport: For Rapid Communication of Neuroscience Research*, 13, 1737–1741.
- Zillman, D. (1994). Cognitive-excitation interdependencies in the escalation of anger and angry aggression. In M. Potegal, & J. Knutson (Eds.), *The dynamics of aggression: Biological and social processes in dyads and groups* (pp. 45–72). Hillsdale, NJ: Erlbaum.
- Ziv, Y., Oppenheim, D., & Sagi-Schwartz, A. (2004). Social information processing in middle childhood: Relations to infant-mother attachment. *Attachment, & Human Development*, 6, 327–348.

Part VI
Lifespan Changes and Gender Differences in Anger

Chapter 19

The Sociological Study of Anger: Basic Social Patterns and Contexts

Scott Schieman

Anger is a highly social emotion. Most people know what it is like to play the part of the angry actor or be a bystander to, or recipient of, somebody else's wrath. It is commonplace to witness the exchange of angry expressions between other individuals in real life, on stage, or in the media. Anger provides drama; rage enlarges it. Anger can sharpen one's critical perspective and creative edge. Its expression can stimulate the lifeless and detached. While there is little doubt that anger can be personally and socially destructive – if it is too intense, enduring, and misdirected – anger can motivate and mobilize efforts against the injustices of everyday life (see Chapter 17).

For sociologists interested in this topic, anger is among the most influential aspects of stress processes and their emotional consequences (Mirowsky & Ross, 2003a; Schieman, 1999). A unique sociological analysis of anger is important because it informs us about the nuances of social life – about social relationships and conditions, the norms and expectations involved in these domains, and the conflicts in society. The sociological study of anger covers a broad range of issues. However, given limited space, I will focus mainly on the key social patterns of anger – or a “social epidemiology” of anger – across the most influential social statuses: gender, age, and social class. That focus sheds light on the core sources of anger provocation that arise in several of the most prominent social contexts, especially in the family/household and workplace. The reductions in everyday anger that go with increasing age and higher social class can be explained by reduced opportunities for provocation. In this effort, I draw upon an array of theoretical perspectives and empirical findings mainly from population-based surveys in the United States.¹

S. Schieman (✉)
University of Toronto, Toronto, ON, Canada
e-mail: scott.schieman@utoronto.ca

¹ I draw upon data from four nationally representative surveys of the United States: the 1990 Work, Family, and Well-Being Survey (WFW), the 1995 Aging and the Sense of Control Survey (ASOC), the 1996 General Social Survey (GSS), and the 2005 Work, Stress, and Health Survey (WSH). These surveys asked questions about the frequency of variants of anger-related feelings and behaviors. Briefly, the WFW survey is a 1990 telephone survey of a national probability sample of U.S. households that contain 2,031 adults who range in age from 18 to 90. Likewise the ASOC survey is a 1995 telephone survey of a national probability sample of U.S. households that contain 2,592 adults who range in age from 18 to 95. See Mirowsky and Ross (2003a, 2003b) for a full description of the WFW and ASOC surveys. The 1996 GSS asks questions about emotions, including items about the frequency, management, and expression of anger, among a sample of 1,460 adults in the United States. See <http://www.icpsr.umich.edu/GSS/> for full details about the GSS sample design and measures. Finally, the 2005 WSH survey is a telephone survey of a national probability sample of U.S. households that contain 1,800 working adults who range in age from 18 to 94. See <http://www.wsh.umd.edu/> for details about the survey.

19.1 Background

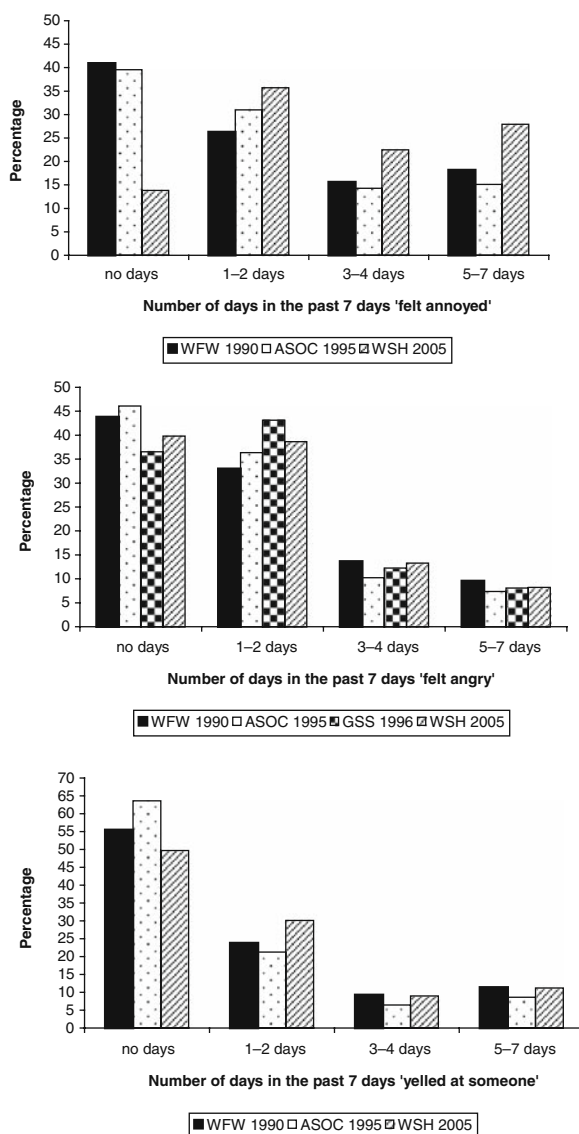
Numerous studies have shown that anger is one of the most frequently reported and recognized emotions (Averill, 1982, 1983; Scherer & Tannenbaum 1986; Scherer & Wallbott, 1994; Shaver, Schwartz, Kirson, & O'Connor, 1987). But what exactly is anger? Given that others in this handbook have provided conceptual specifications of anger, I will only briefly comment on this issue. Anger is typically marked by uncomfortable cognitions and affect and by unique triggers, physiological reactions, expressions, and social consequences (Fehr & Baldwin, 1996). Moreover, anger is often an *interpersonal* event that involves negative appraisal of self or society (Tavris, 1989). Perhaps more than other emotions, anger is one of those “I know it when I feel (or see) it” emotions. Research shows that a majority of adults have little difficulty distinguishing between anger and many other emotions, such as sadness, happiness, and fear (Russell & Fehr, 1994). These distinctions become less clear, however, in the instances of other “*anger-like*” emotions such as frustration, reproach, and resentment (Berkowitz & Harmon-Jones, 2004; Smith & Kirby, 2004).

Anger may be more accurately identified as a process (Schieman, 2006). For example, actors typically conform to an “anger script,” which consists of a prototypical elicitors, physiological sensations, cognitions, actions, and consequences associated with external stimuli (Fehr & Baldwin, 1996). However, the potential varieties of anger-like emotions and anger-related processes engender fuzziness in the conceptualization of anger (Ellsworth & Scherer, 2003; Clore, Ortony, Dienes, & Fujita, 1993). While recognizing the importance of the complex physiological processes, neurological pathways, and facial expressions that distinguish anger (as others in this handbook have described), sociologists have tended to focus attention on individuals’ subjective and perceptual accounts of anger. In particular, many sociologists who investigate anger from a broader population-based approach typically rely on self-reports of anger. For example, in several recent large-scale surveys, anger has been operationalized in terms of feelings that range from mild to more severe (e.g., annoyed, angry, outraged) and behavioral expression (e.g., yelling at someone) (Mirowsky & Ross, 2003a; Schieman, 1999).

What have population-based studies revealed about the frequency of anger among Americans? To answer this question, I draw upon data from four nationally representative surveys of the United States that span from 1990 to 2005. These surveys asked questions about the frequency of variants of anger-related feelings and behaviors. For example, the top panel of Fig. 19.1 shows the frequency of “feeling annoyed in the past 7 days.” In the 1990 WFW and 1995 ASOC surveys, roughly 40% of Americans reported that they did not feel annoyed in the past week. This is quite different from the more recent 2005 WSH survey, which found that only 15% of Americans reported not feeling annoyed at all in the previous week. Moreover, adults in the WSH survey report feeling annoyed much more frequently than participants in the WFW and ASOC surveys. It is noteworthy that more than 25% of WSH participants report feeling annoyed between 5 and 7 days in the past week.

Replication of the same questions (“feel annoyed”) over a 15-year period with different samples in the WFW, ASOC, and WSH surveys allows for potential time series analyses to detect trends. Given these preliminary findings, it seems worthwhile for future investigations to ask: Are Americans annoyed more frequently now than they were in previous years? It should be noted that the WSH survey only interviewed *working* adults, so it is plausible that something about being employed elevates the risk of being annoyed – a point that I will describe in detail later (also see Chapter 24). However, when levels of annoyance are examined only among working adults in the WFW and ASOC surveys, the differences between those surveys and the WSH survey still remain. What has changed in the United States since the early 1990s? The proliferation of loud-speaking users of cell phones in public spaces, increasingly congested traffic, and the constant stream of unpleasant news about terrorism, an unpopular war in Iraq, and global warming, may be contributing to Americans’ feelings of annoyance. Although it is beyond the scope of this chapter to

Fig. 19.1 The Frequency of “feeling annoyed,” “feeling angry,” and “yelling” among Americans



explain the reasons for the apparent increase in annoyed feelings over time, including the possibility of greater honesty in reporting or a more normative acceptance of mild variants of anger, these questions remain ripe for future investigations.

How often do Americans feel *angry*? The patterns for anger across four U.S. surveys are much more consistent than those observed for feelings of annoyance. As the middle panel of Fig. 19.1 illustrates, roughly 37–45% of Americans report that they did not “feel angry” in the past 7 days. Similarly, depending on the survey, 33–42% of Americans report feeling angry once or twice in the past 7 days. Substantially fewer Americans report feeling angry on 3 days or more; roughly 8% report feeling angry on 5–7 days in the past 7 days. Unlike the patterns for annoyance, there is remarkable stability across the span of 1990 and 2005 in overall frequencies of feeling angry. Moreover, unlike

the apparent rising trend from 1990 to 2005 for the milder variant of anger (“feeling annoyed”), there does not appear to be a similar pattern for feeling angry. Here, the divergent patterns for feeling annoyed and angry underscore the value of further investigation of the social causes of the milder anger-variants from what might be conceptualized as a more intense form of anger: *feeling angry*.

In addition to feeling annoyed and angry, some researchers have tracked the frequency of an anger-relevant behavior – *yelling* – and the ways this form of expression contrasts with annoyed and angry *feelings*. Unlike the observed frequencies of annoyance and anger, yelling occurs much less frequently (see the bottom panel of Fig. 19.1). The percentage of Americans who report that they did not yell at all in the past week ranges from 50 (WSH) to 65% (ASOC). However, substantially fewer Americans report that they yelled at someone on ≥ 3 days in the past 7 days. Once again, there is fairly remarkable stability in the frequency of this anger-related behavior across three nationally representative surveys. It seems fairly safe to conclude that Americans do not yell as frequently as they feel annoyed or angry – probably because of the greater intensity and severity of anger that yelling represents as well as its social consequences (also see Chapter 9 for a more complete description of the vocal expression of anger). As I will describe later, yelling is probably more easily identified (compared to annoyance and anger) as evolving directly from conflictive or frustrating social exchanges in the household context.

Having established the basic frequencies of anger-related feelings and behaviors in the population, some sociologists have turned their attention to documenting basic social patterns of anger processes and the reasons for them (Mirowsky & Ross, 2003a; Schieman, 1999, 2000; Simon & Nath, 2004; Stets & Tsushima, 2001). That agenda fits well with a parallel interest understanding the mental health consequences of social stratification found in the sociological study of mental health (McLeod & Nonnemaker, 1999). Pearlin’s (1999) stress process framework offers a useful guide because it underscores the conditions that expose individuals to stressors that might also be viewed as the core sources of anger provocation, including injustice and insult, feelings of betrayal, the sense of inequity or unfairness, being the target of another’s verbal or physical assault, and – especially in the workplace – the incompetence of another person (Berkowitz & Harmon-Jones, 2004; Canary, Spitzberg, & Semic, 1998; Izard, 1977, 1991; Chapter 24). In turn, the systematic patterning of the sites of anger provocation may contribute to social patterns of anger across age, gender, and social class.

19.2 Social Patterns of Anger

In an effort to carve out an agenda for the burgeoning *sociology of emotions* research area, Gordon (1990) proposed that an individual’s social position determines the “type, frequency, and intensity of emotions that will be directed toward him or her or aroused in him or her” (p. 161). That assertion has provided a template for sociological analyses of the ways that core dimensions of social stratification influence emotions, including anger (Ross & Van Willigen, 1996, 1997; Schieman, 1999, 2000). These efforts have contributed to a social “epidemiology of emotions” in which age, gender, and social class emerge as among the most influential dimensions of social stratification on emotion-related processes and outcomes (Smith-Lovin, 1995; Thoits, 1989).

19.2.1 Age and Anger

A central question in the sociological study of anger has been “Do older adults report less anger than their younger peers?” There is a sound theoretical rationale and solid empirical evidence for

suggesting that average levels of anger are lower among older adults (Carstensen, Pasupathi, Mayr, & Nesselrode, 2000; Mirowsky & Ross, 2003a; Schieman, 1999). For example, data from the 1996 GSS indicate that average levels of feeling mad, angry, and outraged tend to decrease across age categories (panel A of Fig. 19.2). Likewise, similar patterns are observed in the more recent 2005 WSH survey such that average levels of feeling annoyed, angry, yelling, and losing one's temper generally tend to fall with increasing levels of age, especially after the thirties (panel B of Fig. 19.2). Moreover, there is a strong negative association between age and overall indices of anger in the GSS

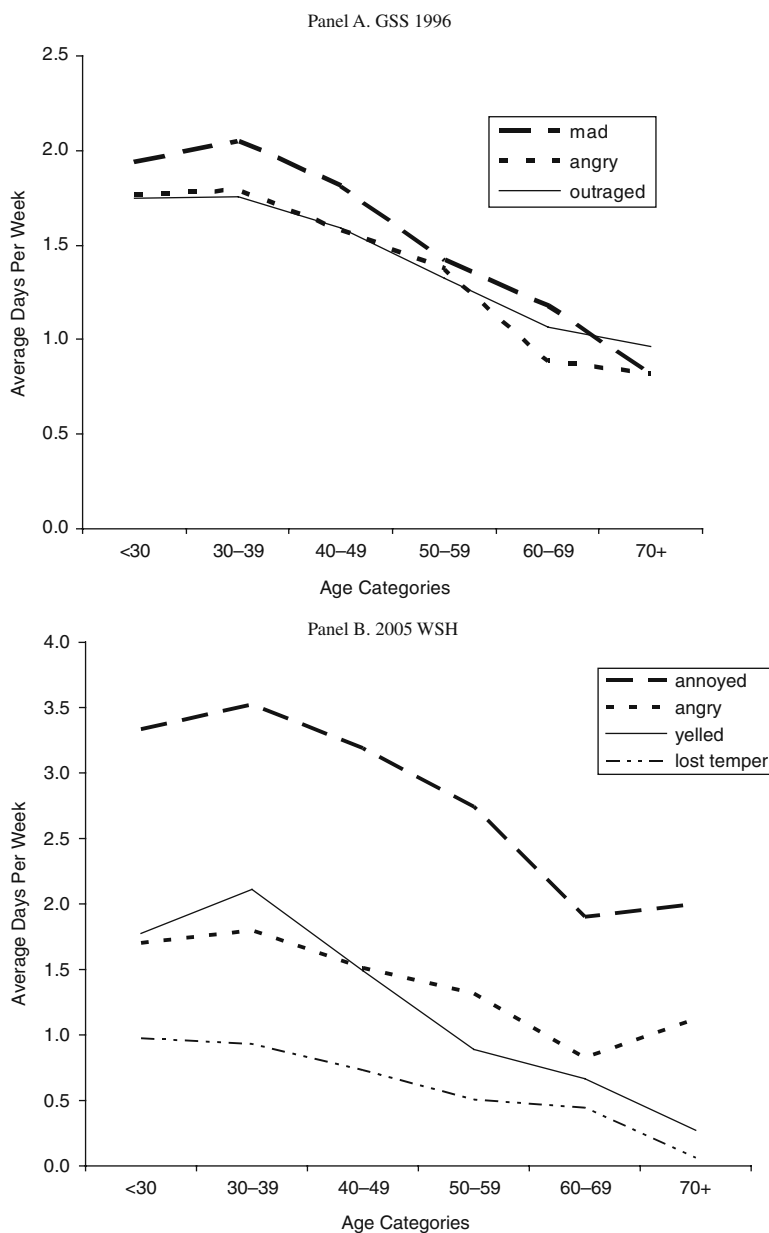


Fig. 19.2 Average levels of variants of anger across age

($\beta = -0.20$, $p < 0.001$) and WSH surveys ($\beta = -0.22$, $p < 0.001$).² These observed age patterns in anger are highly consistent with those observed in the 1990 WFW and 1995 ASOC surveys (see Mirowsky & Ross, 2003a, p. 114; Ross & Van Willigen, 1997, p. 287), as well as a survey of residents in Southwestern Ontario, Canada (Schieman, 1999). The negative association between age and anger persists into late-life, as observed in a 2000–2001 community survey of White and African-American adults aged 65 and older in the Washington, DC, metropolitan area (Schieman & Meersman, 2004).

It appears that older people experience (or report) anger less frequently than their younger peers, but are there age differences in other anger-related processes? Analyses of the 1996 GSS indicate that older adults are significantly less likely to agree with the statement “when I am angry I let people know” (Schieman, 2000). Moreover, when angered, older adults are *less likely* than younger adults to do the following: talk to the target of their anger, try to think differently about the anger-provoking situation, try to change the situation by doing something, drink alcohol or take pills, or talk to someone else about the anger. Also, when describing a specific anger-provoking episode, older people report less intense anger but are no different than their younger peers in the average duration of the angry feelings or the perception about the appropriateness of their response to the anger (Schieman, 2000; Simon & Nath, 2004; Stets & Tsushima, 2001). While these basic social patterns underscore age differences in anger-related processes, future research should seek to rule out the influence of age-related biases in reporting.

19.2.1.1 External Versus Internal Factors

Given the well-established negative association between age and the frequency of anger described above, sociological analyses have turned to explaining the reasons for that pattern. Age marks social status, stratification, and life course position. Thus, it also generates systematic patterns of emotional processes associated with the relationships and stressors embedded in core social role arrangements. Here, stress process and life course theories offer clues about age patterns in anger because they highlight age-related role stressors (Pearlin & Skaff, 1996; Schieman, Van Gundy, & Taylor, 2001). The central thesis is that age-based variations in the structural and subjective organization of peoples’ lives contribute to systematic age patterns in exposures to the sites of anger provocation (Schieman, 2006).

Research across societies and cultures reveals that most emotion-related experiences (negative and positive) are typically linked to relationships in work or family roles (Scherer & Tannenbaum, 1986; Scherer, Wallbott, & Summerfield, 1986). Sociologists have theorized the centrality of power-status dynamics in these social roles and their differential influence on emotions (Kemper, 2006). The most *intense* experiences of many different emotions are likely to occur in close, intimate relationships because individuals tend to be highly invested in those relations (Carstensen, Graff, Levenson, & Gottman, 1996). Moreover, the family context is ripe with opportunities for stress exposure (Pearlin & Turner, 1987). As Chapter 29 and Chapter 30 suggest, anger in the family is

² The 1996 GSS asked: “On how many days in the past 7 days have you felt. . .” (1) “mad at someone or something,” (2) “angry,” and (3) “outraged.” When these three items are averaged to create an index of anger, the overall mean is 1.55 days. A principal component factor analysis shows that the “mad,” “angry,” and “outraged” items each loads highly on one construct (0.69, 0.84, and 0.84, respectively) and the alpha reliability coefficient for the index is 0.85. The 2005 WSH survey assessed anger with four questions: “On how many days in the past 7 days have you. . .” (1) “felt annoyed,” (2) “felt angry,” (3) “yelled at someone or something,” and (4) “lost your temper”? When these four items are averaged to create an index, the mean is 1.67 days. A factor analysis shows that each of these items load highly on one construct (loadings from 0.74 to 0.82) and the reliability coefficient for the index is 0.76. Analyses of curvilinear age-anger patterns do not yield a significantly better fit than a linear model.

particularly salient in partner and parent–child relationships. Snyder describes the role of anger in the etiology and development of children’s problem behaviors in response to parental actions. In particular, parents’ nagging, criticism, and other low intensity/low anger aversive approaches to shaping children’s behavior is likely to evoke reciprocal opposition and anger in children.

Taken together, these ideas imply that family/household and work conditions – individually and in conjunction with each other – create numerous occasions for anger-related experiences that may be differentially distributed across the age span. For example, young adulthood is a time of budding intimate relationships, family formation, and the establishment of a household. However, increasing age modifies these processes such that older individuals are less likely to be in a cohabiting arrangement, and more likely to encounter the death of friends and relatives, the exit of children from the household, and the death of a spouse or partner. Likewise, older adults are less likely to have children or teenagers residing in the household – a central source of anger in everyday life (Dix, 1991; Schieman, 1999). In addition to fewer exposures to anger elicitors associated with children, older adults are less likely to have parental responsibilities that absorb their time and attention, which in turn may reduce the feeling of being rushed for time (Bianchi, Robinson, & Milkie, 2006; Mattingly & Sayer, 2006). There is also evidence that older adults experience lower levels of other social causes of anger, including economic hardship (Mirowsky & Ross, 2003a), work–home conflict (Mennino, Rubin, & Brayfield, 2005), job dissatisfaction (Kalleberg & Loscocco, 1983), and interpersonal conflict in the workplace (Schieman & Reid, 2008).

Age variations in psychosocial resources may also influence age patterns in anger. For example, the “age as maturity” view implies that advancing age equips individuals with the psychosocial tools (i.e., the sense of trust) needed to develop and sustain stable intimate relationships with less conflict (Mirowsky & Ross, 1992). According to Birditt and Fingerman (2005), older adults tend to be better at regulating their behavioral responses to interpersonal conflicts than their younger peers. These ideas are consistent with Carstensen’s (1992) “social–emotional selectivity” theory, which predicts that adults in late-life are more effective than younger adults at conserving their emotions, possess more self-control, and a greater capacity for tolerance, even in circumstances that present conflict. Collectively, these views about age-related social conditions generate the following claim: Age should be associated negatively with anger partly because older people are less likely to have children under the age of 18 residing in the household, less likely to have to arrange and deal with childcare responsibilities, less likely to feel rushed for time in everyday life, and, consequently, less likely to be exposed to the stress of home-to-work conflict. These conditions are each associated positively with anger; by extension, analyses that take these interrelationships into account should help to explain some of the negative age–anger association. Other conditions, such as greater job satisfaction, greater access to nonroutine work, a greater generalized sense of trust in others, and higher levels of religiosity should also contribute to less anger among older adults (Schieman, 2006; Schieman et al., 2001).

Although these ideas have been generally supported in prior studies (Mirowsky & Ross, 2003a; Schieman, 1999), here I reexamine the question with data from the 2005 WSH survey. As shown in model 1 of Table 19.1, age is associated positively with an anger index net of sex, race, and education ($\beta = -0.22$, $p < 0.001$). Regression analyses that adjust for each of the family, household, work, and psychosocial conditions described above fully account for the negative age–anger association.³ Specifically, these statistical adjustments reduce the size of the standardized age coefficient from -0.223 to -0.048 and that effect is no longer statistically significant at the 0.05 level

³ There is insufficient space to provide details about each measure in Table 19.1. All items, response choices, and scale properties are available upon request. Broadly speaking, the items and measures are based on those used in numerous other studies. For example, the sense of trust index is comprised of three items that have appeared in the General Social Survey since 1972.

Table 19.1 Regression of Anger on Age and Explanatory Conditions

Variables	Model 1	Model 2
Age	−0.223***	−0.048
Women = 1	0.149***	0.085***
Education	−0.107***	−0.053*
African-American ^a	0.010	0.004
Other race ^a	−0.057*	−0.046*
Number of people in household	—	0.064**
Primarily responsible for childcare	—	0.073**
Divorced/separated ^b	—	−0.040
Widowed ^b	—	−0.042*
Never married ^b	—	−0.033
In a cohabiting relationship	—	0.059*
Economic hardship	—	0.138***
Feeling rushed for time	—	0.175***
Negative life events	—	0.058**
Home-to-work conflict	—	0.093***
Work-to-home conflict	—	0.066**
Interpersonal conflict at work	—	0.100***
Nonroutine work	—	−0.049*
Job satisfaction	—	−0.098***
Sense of trust	—	−0.098***
Religiosity	—	−0.076***
Constant	3.018	0.342
R-Square	0.090	0.308

* $p < 0.05$; ** $p < 0.01$; *** $p < 0.001$ (two-tailed test).

Note: Standardized regression coefficients are shown in the table.

^aCompared to Whites.

^bCompared to married.

(see model 2 of Table 19.1). Although statements about the *causal* associations in cross-sectional analyses are not definitive here – especially in the linkages between anger and the family/household and work conditions – these patterns do provide preliminary evidence that can guide future investigation about the reasons why older people report less anger. For example, the three most influential reasons are older adults' tendency for lower levels of feeling rushed for time, their lower levels of economic hardship, and their less frequent exposure to interpersonal conflict in the workplace.

While each of those conditions deserves greater attention, I briefly review only the most influential: *feeling rushed for time*. The sense that one has insufficient time to deal with demands, expectations, and responsibilities in core social roles is likely to be one of the most frustrating, chronic conditions of everyday life (Mattingly & Sayer, 2006). Individuals who report that they *always* feel rushed for time also report significantly more frequent feelings of annoyance, anger, and are more likely to yell or lose their temper (see Fig. 19.3). Clinically, the experience of time pressure is one component of the anger-associated “type A” personality pattern, as reviewed in Chapter 25. This is also a critical – but understudied – social pattern, especially in the context of age differences: Older adults are *less likely* to feel rushed for time. By extension, these age-based patterns in feeling rushed for time help explain why older adults tend to report less anger. An important next step for researchers is to document the conditions that contribute to these processes, especially in household and workplace contexts. Moreover, establishing these patterns over time in longitudinal designs will also help delineate the causal mechanisms and directions involved.

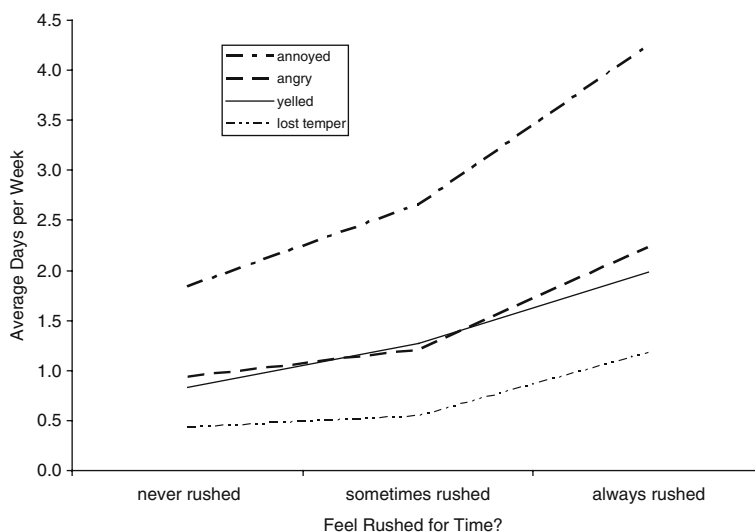


Fig. 19.3 Feeling rushed for time and average levels of anger (WSH 2005)

19.2.2 Gender and Anger

Do women and men differ in their experiences of anger? Gender stereotypes of emotions imply that anger is a more “acceptable emotion” for men (Jansz, 2000), partly because it is often viewed as a masculine emotion linked with status and power (Mirowsky & Ross, 1995, see Chapter 21 for arguments about how anger is particularly adaptive for men). Although research shows that individuals’ *perceptions* of gender and emotion reinforce the notion that women experience and express emotions more often and intensely than men (Johnson & Shulman, 1988), anger appears to be *the exception*. Adults are more likely to perceive men as expressing anger more frequently (Fabes & Martin, 1991). Strachan and Dutton (1992:1721) contend that “behavioral expectations based on gender encompass many aspects of interpersonal relationships including rules that govern gender-appropriate affect.” By extension, display rules dictate that if women get angry they are expected *not* to show it (Brody, 1999), perhaps out of a fear that anger expression will exact a cost to their self and relationships (Egerton, 1988; Hess, Adams, & Kleck, 2005). If women *do* express anger, they risk being labeled “hostile,” “neurotic,” “unladylike,” or worse (Sharkin, 1993; Tavris, 1989). Moreover, male leaders are rated as less effective when they express sadness, while female leaders are rated as less effective if they express sadness *or* anger (Lewis, 2000).

Collectively, these ideas suggest it is reasonable to suspect that men should, on average, report more frequent and intense anger than women, and they should be more likely to express it. Evidence from population-based surveys, however, is inconclusive. Analyses of the 1996 GSS did not find gender differences in the frequency of feeling mad, angry, or outraged (Schieman, 1999), while other studies document that women tend to report a greater frequency of anger (Mirowsky & Ross, 2003a; Strachan & Dutton, 1992). The 2005 WSH survey finds that, compared to men, women report a higher average frequency of feeling annoyed, angry, yelling, and they are more likely to lose their temper (all differences significant at $p < 0.001$; see Fig. 19.4).

The focus on expression is essential. Like the WSH survey, evidence in the 1996 ASOC survey shows that women are generally more likely than men to express their anger by yelling (Mirowsky & Ross, 1995, 2003a). Likewise, analyses of the 1996 GSS indicate that 70.1% of women compared to

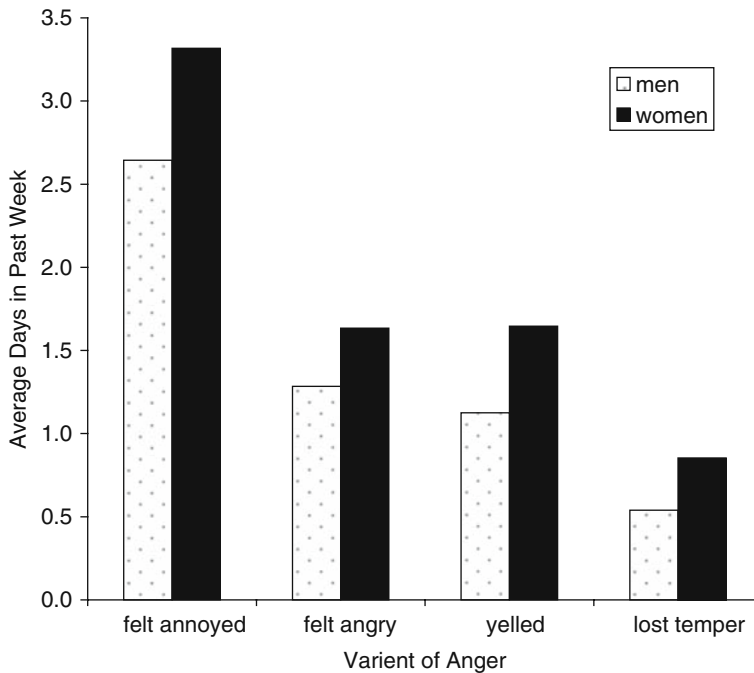


Fig. 19.4 Average levels of variants of anger by gender (WSH 2005)

64.4% of men agree or strongly agree with the statement “When I’m angry I let people know” ($\chi^2 = 4.99, p < 0.05$). While these social patterns appear somewhat antithetical to the nurturing stereotype of women, it is important to note that studies consistently find that men are significantly more likely to agree with the more general statement about emotional reserve: “I keep my emotions to myself” (Mirowsky & Ross, 1995, 2003a). For example, in the 1996 GSS, 52.3% of men compared to 43.2% of women agree or strongly agree with that statement ($\chi^2 = 5.56, p < 0.05$). Gender differences in emotional reserve in the 2005 WSH survey are even greater: 62.7% of men agree or strongly agree with the emotional reserve statement compared to 47.1% of women ($\chi^2 = 41.73, p < 0.001$).

Another analysis of the 1996 GSS found that, once angered, women think about the anger more, talk to the person they feel angry with more, and take longer to stop feeling angry (Schieman, 2000). In addition, intensity is another key dimension of the anger experience (for example, see Chapter 12). Affect intensity “refers to one’s response intensity to a given level of emotion-provoking stimulation” (Fujita, Diener, & Sandvik, 1991:428). Common views predict that, when angered, men should report more intense angry feelings than women do. Although people tend to *perceive* that men’s anger is more intense (Kring, 2000), the 1996 GSS data indicate that women tend to report more intense anger than men (Simon & Nath, 2004; Stets & Tsushima, 2001). This is consistent with other findings that women feel negative emotions more intensely (Fujita et al., 1991), including anger (Brody, 1999; Fehr & Baldwin, 1996). Cross-national data, including surveys in the United States, suggest that men tend to see anger as taking control of a situation while women tend to experience anger as losing control of themselves (e.g., Astin et al., 2003). However, some evidence suggests that women are more likely than men to suppress anger (Haynes & Feinleib, 1980), while others fail to detect differences in suppression or expression (Kopper & Epperson, 1991; Milovchevich, Howells, Drew, & Day, 2001). By contrast, men tend to be more likely to verbally or physically assault the target of anger (Deffenbacher, Oetting, Lynch, & Morris, 1996).

Taken together – despite recent claims that “women and men get angry with the same intensity and frequency” (Mayne & Ambrose, 1999:356) – the empirical evidence seems to yield indefinite conclusions about gender differences (Kring, 2000, for an alternative view, see Chapter 20). People have many opinions about gender and patterns of anger – but the evidence does not consistently corroborate those opinions. The lack of definitive findings about gender differences in anger processes may be due to variations across work and household contexts. The gendered nature of work and family roles and the differential relevance of these role identities for self-esteem and potency might foster different gender experiences of anger (Schieman, 2000; Simon & Nath, 2004; Stets & Tsushima, 2001). The 1996 GSS asked participants to recall a specific episode of anger in the past month and identify the domain in which it occurred. Women are significantly more likely than men to report that the anger occurred in the family domain (Schieman, 2000). Specifically, 40% of women reported feeling anger in the family context compared to only 19% of men ($\chi^2 = 48.91$, $p < 0.001$). Moreover, the same percentages of men (19%) report family-related anger regardless of whether they are currently employed or not, while 50% of non-working women report family-related anger compared to 34% of working women.

Outside of the workplace, individuals often spend a substantial portion of time involved in child-care activities – especially women (Bianchi, et al., 2006). Thus, the parent role contains many opportunities for a range of positive and negative emotions; anger is one of the most prominent negative ones (Carpenter & Halberstadt, 1996; Scherer & Tannenbaum, 1986; Chapter 29). Dix (1991) has observed that “average parents report high levels of anger with their children, the need to engage in techniques to control their anger, and fear that they will at some time lose control and harm their children” (p. 3). In analyses of the 2005 WSH survey, I observe that the number of children in the household is associated positively with scores on the anger index – but, as the top panel of Fig. 19.5 illustrates, this association is significantly stronger among women, especially those in full-time jobs (i.e., approximately 40 hours per week). Separate analysis of each individual anger-related item in the anger index reveals that these patterns are particular strong for “yelling,” as shown in the bottom panel of Fig. 19.5. Moreover, the intersection of home and work lives is potentially conflictive for working women with children. For example, analysis of the 2005 WSH survey documents that home-to-work conflict is associated more strongly with anger among working women – especially those in households with children. Collectively, these patterns demonstrate that family/household conditions cultivate the seeds of anger-related processes in different ways for women and men. Moreover, given these findings and those underscoring the importance of feeling rushed for time, the potential influence of the home-work interface on anger-related processes and outcomes deserves much more attention in population-based analyses.

19.2.3 Social Class and Anger

The ways that social stratification and inequality influence emotional experience represents a long-standing interest in sociology, especially the sociological study of stress (Pearlin, 1999). Dimensions of social class – such as education, income, and work conditions – reflect sources of status, power, and resources that connect people to social organization and culture; in turn, these socioeconomic conditions influence emotional life (Mirowsky & Ross, 2003a, 2003b; Thoits, 1989). For example, while it is well documented that education is associated negatively with other emotional outcomes like depression and anxiety (McLeod & Nonnemaker, 1999), the effects of education on anger are much more complicated (Schieman, 2000). Studies of education-based differences in the frequency of anger yield mixed results. In the 1990 WFW survey, education is *unrelated* to an index of anger

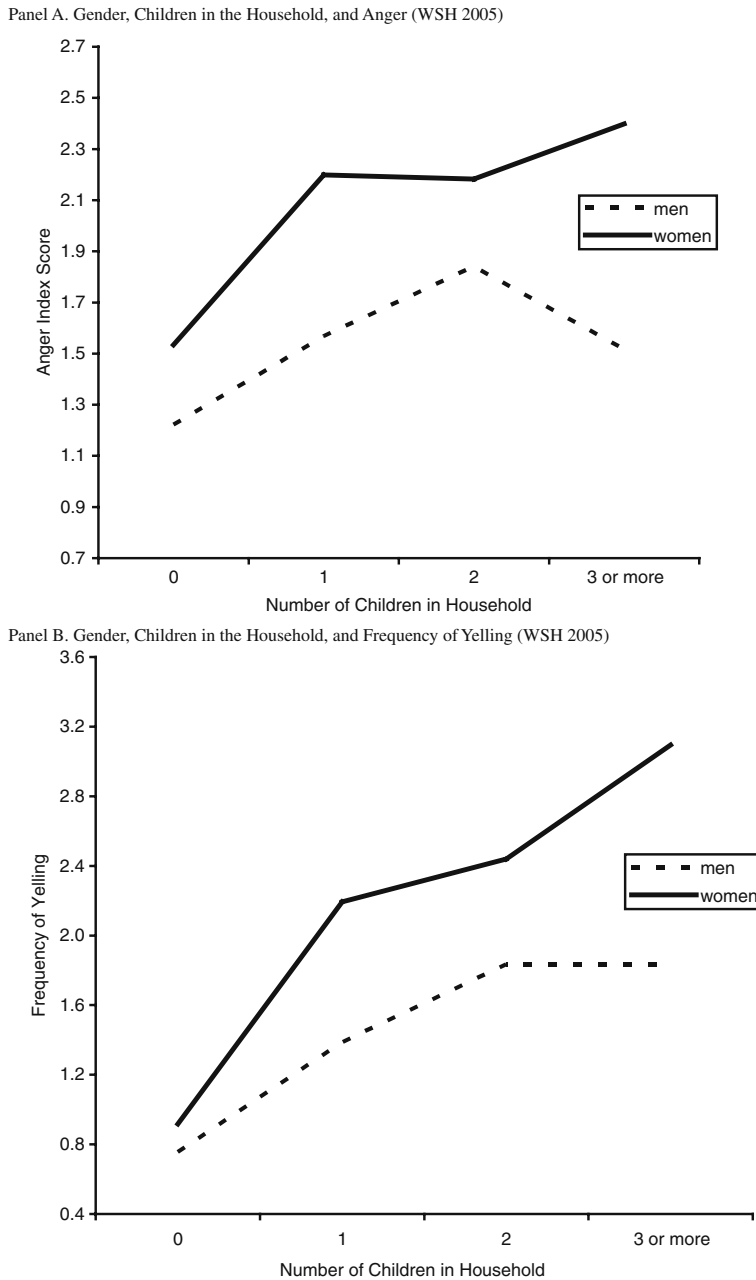


Fig. 19.5 Gender, children in the household, and the frequency of anger and yelling

(Mirowsky & Ross, 1995), but it is related *negatively* to yelling. By contrast, both the 1995 ASOC survey (Ross & Van Willigen, 1996) and the 2005 WSH survey find that education is associated negatively with the frequency of the variants of anger. The top panel of Fig. 19.6 illustrates these patterns in the WSH survey. The lowest levels of feeling annoyed, angry, yelling, and losing one's

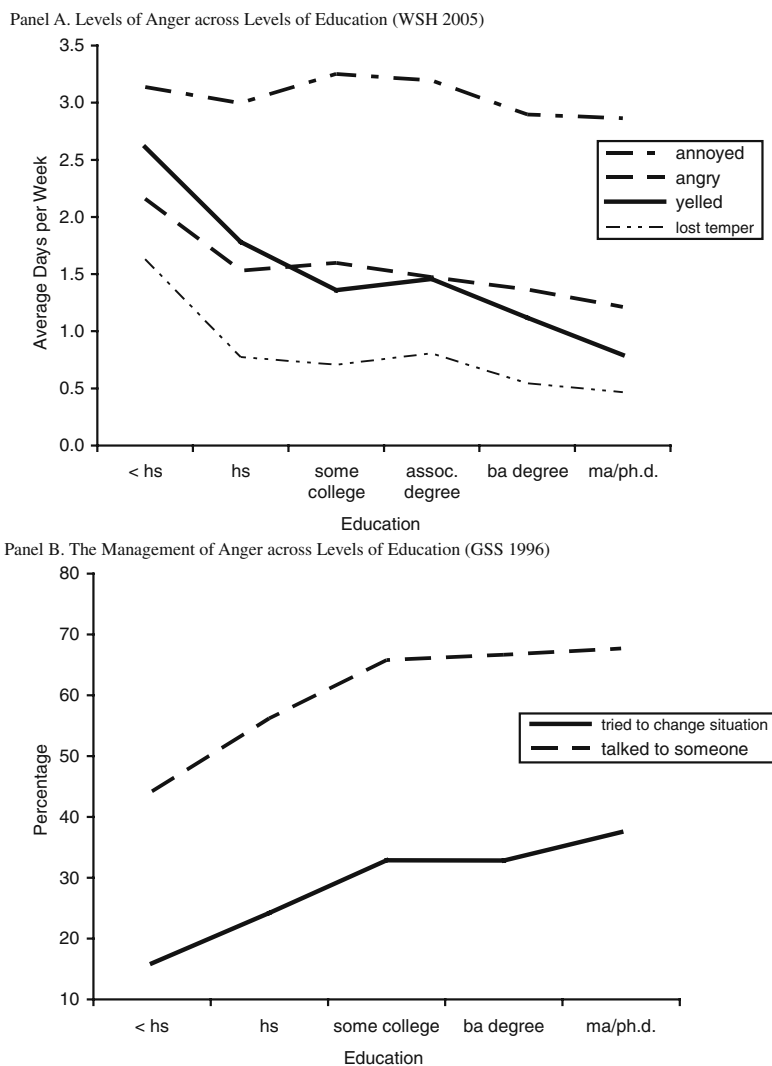


Fig. 19.6 Education and anger

temper are observed among those with advanced education. It is important to underscore, however, that the strongest patterns are observed for yelling and losing one's temper, while education-based differences in feeling annoyed are much smaller. Table 19.1 shows the negative and significant effect of education on the overall anger index.⁴

⁴ For ease of presentation I analyze education as an ordinal-like continuous variable. Separate analyses that distinguish between categorical versus continuous measures of education reveals similar results. This is due to the linear association between education and the anger index. These analyses provide a starting point for investigation into the education-based patterns of anger. Future research should examine education-based differences in detail – drawing distinctions between degree categories, other credentials, and areas of emphasis among those with college or postgraduate degrees, as well as distinctions in its effect on the different forms of anger.

What explains the negative association between education and anger? Adjustment for the conditions shown in model 2 substantially reduces the education coefficient although it remains statistically significant. This reduction is attributable to well-educated individuals' lower levels of economic hardship ($r = -0.26, p < 0.001$), their greater sense of trust ($r = 0.25, p < 0.001$), and the fact that they tend to reside in households with fewer people ($r = -0.078, p < 0.01$). Collectively, these conditions explain a substantial portion of the negative education–anger association. The 1996 GSS indicates that the well-educated are less likely than their less-educated peers to report anger that is provoked in the family context ($OR = 0.911, p < 0.001$) and more likely to report anger that is provoked in the work context ($OR = 1.072, p < 0.01$).⁵

Collectively, the education–anger patterns are generally consistent with theoretical views of education as a component of socioeconomic status that has consequences for exposure to important stressors and as a source of psychosocial resources (McLeod & Nonnemaker, 1999; Ross & Van Willigen, 1997). The “resource” dimension of education is especially relevant when we examine the ways that individuals *act* once they are angry. That is, does education influence the management of anger? As the bottom panel of Fig. 19.6 illustrates, data from the 1996 GSS indicate that the well-educated are much more likely to be proactive by trying to change the situation that caused the anger and by attempting to think differently about the anger-provoking situation (Schieman, 2000; Simon & Nath, 2004). When anger-provoking situations arise, the well-educated are more likely to possess flexible cognitive skills that illuminate alternative sides of an issue – an ability that determines anger's course (Mirowsky & Ross, 2003b; Tavris, 1989).

Income represents another core dimension of social stratification and an important source of inequality – especially as it relates to subjective assessments of economic hardship, satisfaction with financial conditions, and perceived inequity in personal earnings (i.e., “feeling underpaid for the work you do”). Before exploring these conditions, however, it is worth noting that income itself is associated negatively with anger. As Fig. 19.7 illustrates, the 2005 WSH data reveal lower average levels of anger among individuals in the upper-income strata; although there is a negative income–anger pattern of association, there is an unusual (and small) “blip” in level of anger among individuals with earnings between \$60,000 and \$79,000. The reasons for this pattern are beyond the scope of this chapter, but there is little question that a potential nonlinear pattern in the income–anger association and the reasons for it deserves closer scrutiny.

The status inequality associated with class-based stratification may be particularly relevant for emotional inequality. For example, the perceived hardships related to economic deficiencies probably generate chronic feelings of frustration and anger. In family contexts, money issues are especially relevant for anger (Carpenter & Halberstadt, 1996). Money may not buy happiness, but financial deficiencies that thwart the purchase of basic needs likely generates aggravation and discontent. Thus, the lack of financial resources may become a chronic drag on everyday life and a central wellspring of anger. Recognizing this, sociologists of mental health have established that economic hardship is one of the most important determinants of psychological well-being (Mirowsky & Ross, 2003a, 2003b). The fact that economic hardship is differentially distributed across core social statuses such as age and gender has also underscored its role as an explanation for gender and age differences in distress. Moreover, the *effect* of economic hardship on anger may differ across these social statuses. For example, evidence from the 2005 WSH survey supports that claim. Figure 19.8 illustrates that the positive association between economic hardship and anger is stronger among women (panel a)

⁵ In these analyses, education is examined as a continuous variable and logistic regression models adjust for gender, age, marital status, household composition, and employment status.

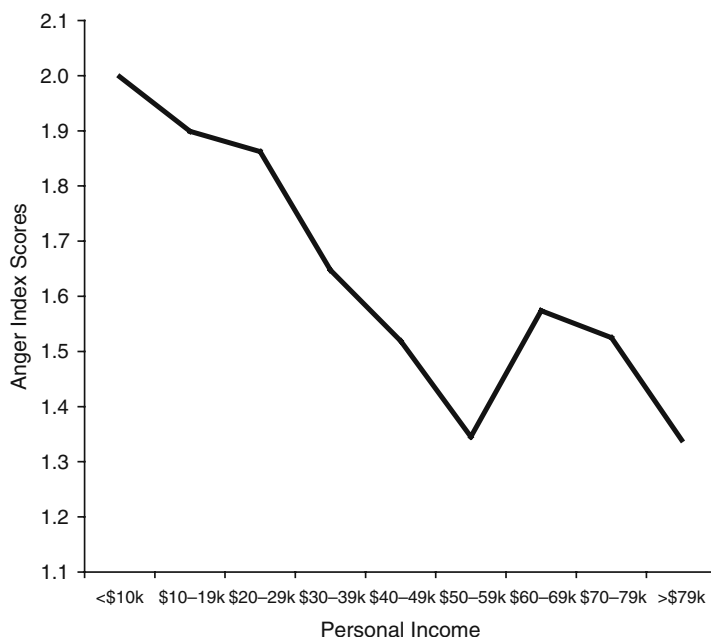


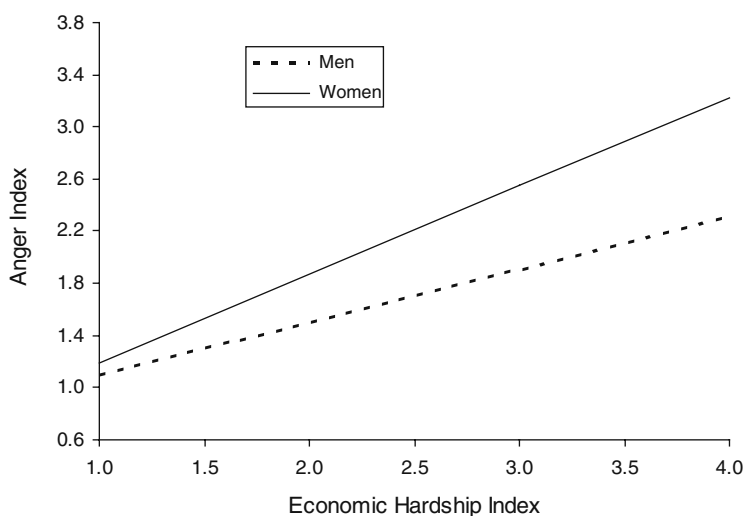
Fig. 19.7 Average levels of anger across levels of income (WSH 2005)

and younger adults (panel b); both of these status-contingent effects are statistically significant at the $p < 0.01$ level.⁶

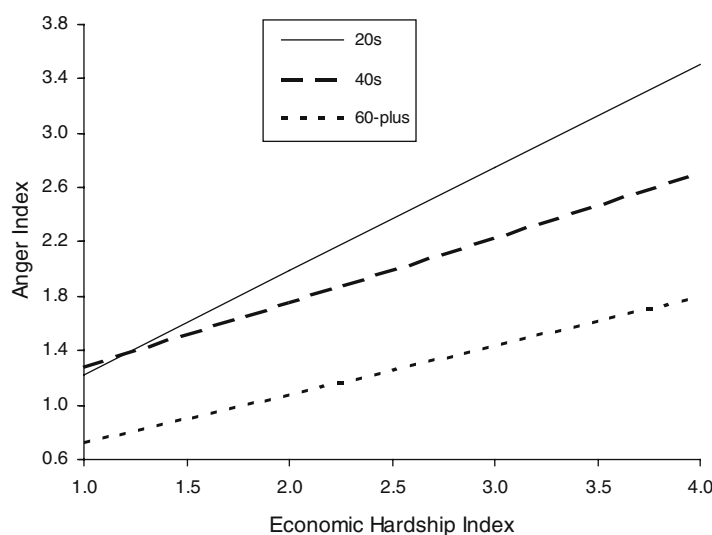
Like economic hardship, perceived inequity is a potent source of disgruntlement. According to equity theory, perceptions of inequality cultivate feelings of frustration and anger (Ross & Van Willigen, 1996). Receiving less than one feels that he or she deserves is an unfair or unjust state of affairs (deCarufel, 1979). Feeling underpaid probably yields some of the highest levels of anger, especially the type of anger that persists over time and contributes to both episodic anger *and* angry moods that may contribute to other mental health outcomes like depression. There are likely important connections between feeling underpaid and a sense of injustice and righteous anger in the workplace (see Chapter 24 for a detailed analysis of the interpersonal and emotional dynamics associated with the sense of injustice). This is an important path for research because most prior evidence about the effects of feeling underpaid focus primarily on depression not anger (Mirowsky and Ross 2003a). Moreover, the 2005 WSH survey indicates that the *majority* of workers report feeling underpaid (21% feel “very underpaid” and 34% feel “underpaid a little”). Compared to those who feel adequately paid for the work that they do, feeling underpaid is associated positively with scores on the anger index ($OR = 1.86$, $p < 0.001$). More research is needed, however, to determine potential personal and social contingencies in the effects of feeling underpaid on anger. For example, is the association different for men versus women, younger versus older adults, the well-educated versus

⁶ Economic hardship is measured by asking the following: “During the last year, how often did you . . .”: “have trouble paying the bills?” “not have enough money to buy food, clothes, or other household good?” and “not have enough money to pay for medical care?” Response choices are (1) “never,” (2) “rarely,” (3) “sometimes,” and (4) “frequently.” A fourth item asks: “How do your finances usually work out by the end of the month? Do you have . . .”: (1) “a lot of money left over,” (2) “a little money left over,” (3) “just enough to make ends meet,” or (4) “not enough to make ends meet?” The four items are averaged to create the hardship index ($\alpha = 0.82$).

Panel A. Gender Differences



Panel B. Age Differences

**Fig. 19.8** Economic hardship and anger by gender and age (WSH 2005)

those with less formal education, and those in different income categories? Is it stronger among individuals who are also experiencing economic hardship? Evidence about these contingencies will enhance knowledge about the differential potency of perceived economic inequality. It is also important to establish greater clarity in the causal ordering in these processes. Currently, in cross-sectional designs, we do not know if perceived financial inequity a cause or consequence of anger. Although theory predicts anger is an outcome, longitudinal evidence in population studies will help solidify knowledge on this topic.

19.3 Conclusion

Social contexts influence the conceptual parameters and processes associated with any emotion (Thoits, 1989); anger is no exception. As others have observed in this handbook, uncomfortable cognitions and affect, physiological reactions or bodily sensations, and expressions or gestures are elements of anger. However, language and the contextual factors surrounding situational stimuli provide the meanings and cultural labels that enable actors to identify an emotional experience as “anger.” Therefore, even if researchers in the burgeoning area of affective neuroscience discover that dimensions of anger processes are the product of genetic heritage and preprogrammed into human brains, the social situations and arrangements of individuals’ lives will remain relevant because they provide the contexts that provoke or confine anger in patterned, systematic ways. Social conditions cause, mediate, and modify the evocation and expression of “hardwired” emotions (see Chapter 8 and Chapter 10). Sociology can help document the social patterns of anger and the conditions that influence the activation, course, expression, and management of anger *as a process*.

References

- Averill, J. R. (1982). *Anger and aggression: an essay on emotion*. New York: Springer-Verlag.
- Averill, J. R. (1983). Studies on anger and aggression: implications for theories of emotion. *American Psychologist*, 38, 1145–1160.
- Berkowitz, L., & Harmon-Jones, E. (2004). Toward an understanding of the determinants of anger. *Emotion*, 2, 107–130.
- Bianchi, S. M., Robinson, J. P., & Milkie, M. A. (2006). *Changing rhythms of American family life*. New York: Russell Sage.
- Birditt, K. S., & Fingerman, K. L. (2005). Do we get better at picking our battles? Age group differences in descriptions of behavioral reactions to interpersonal tensions. *Journals of Gerontology: Psychological Sciences*, 60, P121–P128.
- Brody, L. R. (1999). *Gender, emotion, and the family*. Cambridge, MA: Harvard University Press.
- Canary, D. J., Spitzberg, B. H., & Semic, B. A. (1998). The experience and expression of anger in interpersonal settings. In P. A. Andersen & L. K. Guerrero (Eds.), *Handbook of communication and emotion: research, theory, applications, and contexts* (pp. 189–213). San Diego: Academic Press.
- Carpenter, S., & Halberstadt, A. G. (1996). What makes people angry? Laypersons’ and psychologists’ categorizations of anger in the family. *Cognition and Emotion*, 10, 627–656.
- Carstensen, L. L. (1992). Social and emotional patterns in adulthood: support for socioemotional selectivity theory. *Psychology and Aging*, 7, 331–338.
- Carstensen, L. L., Graff, J., Levenson, R. W., & Gottman, J. M. (1996). Affect in intimate relationships. In C. Magai & S. H. McFadden (Eds.), *Handbook of emotion, adult development, and aging* (pp. 227–248). San Diego, CA: Academic Press.
- Carstensen, L. L., Pasupathi, M., Mayr, U., & Nesselroade, J. R. (2000). Emotional experience in everyday life across the adult life span. *Journal of Personality and Social Psychology*, 79, 644–655.
- Clare, G. L., Ortony, A., Dienes, B., & Fujita, F. (1993). Where does anger dwell? In R. S. Wyer Jr. & T. K. Srull (Eds.), *Advances in social cognition: perspectives on anger and emotion*, vol. 6 (pp. 57–87). Hillsdale, NJ: Lawrence Erlbaum Associates.
- deCarufel, A. (1979). Pay secrecy, social comparison, and relative deprivation in organizations. In J. Olson, P. Herman, & M. Zanna (Eds.), *Relative deprivation and social comparison: the Ontario symposium*, (Vol. 4, pp. 181–200). Hillsdale, NJ: Lawrence Erlbaum Associates.
- Deffenbacher, J. L., Oetting, E. R., Lynch, R. S., & Morris, C. A. (1996). The expression of anger and its consequences. *Behavior Research and Therapy*, 34, 575–590.
- Dix, T. (1991). The affective organization of parenting. *Psychological Bulletin*, 110, 3–25.
- Egerton, M. (1988). Passionate women and passionate men: sex differences in accounting for angry and weeping episodes. *British Journal of Social Psychology*, 27, 51–66.
- Ellsworth, P. C., & Scherer, K. R. (2003). Appraisal processes in emotion. In R. J. Davidson, H. Goldsmith, & K. R. Scherer (Eds.), *Handbook of the affective sciences* (pp. 592–595). New York and Oxford, England: Oxford University Press.

- Fabes, R. A., & Martin, C. L. (1991). Gender and age stereotypes of emotionality. *Personality and Social Psychology Bulletin*, 17, 532–540.
- Fehr, B., & Baldwin, M. (1996). Prototype and script analyses of lay people's knowledge of anger. In G. J. O. Fletcher & J. Fitness (Eds.), *Knowledge structures in close relationships: a social psychological analysis* (pp. 219–246). Hillsdale, NJ: Erlbaum.
- Fujita, F., Diener, E., & Sandvik, E. (1991). Gender differences in negative affect and well-being: the case for emotional intensity. *Journal of Personality and Social Psychology*, 61, 427–434.
- Gordon, S. L. (1990). Social structural effects on emotions. In T. D. Kemper (Ed.), *Research agendas in the sociology of emotions* (pp. 145–179). Albany: State University of New York Press.
- Haynes, S. G., & Feinleib, M. (1980). Women, work, and coronary heart disease: prospective findings from the Framingham Heart Study. *American Journal of Public Health*, 70, 133–141.
- Hess, U., Adams, R. B., Jr., & Kleck, R. E. (2005). Who may frown and who should smile? dominance, affiliation, and the display of happiness and anger. *Cognition and Emotion*, 19, 515–536.
- Izard, C. E. (1977). *Human emotions*. New York: Plenum Press.
- Izard, C. E. (1991). *The psychology of emotions*. New York: Plenum.
- Jansz, J. (2000). Masculine identity and restrictive emotionality. In A. H. Fischer (Ed.), *Gender and emotion: social psychological perspectives* (pp. 166–186). Cambridge, UK: Cambridge University Press.
- Johnson, J. T., & Shulman, G. A. (1988). More alike than meets the eye: perceived gender differences in subjective experience and its display. *Sex Roles*, 19, 67–79.
- Kalleberg, A. L., & Loscocco, K. A. (1983). Aging, values, and rewards: Explaining age differences in job satisfaction. *American Sociological Review*, 48, 78–90.
- Kemper, T. D. (2006). Power and status and the power-status theory of emotions. In J. E. Stets & J. H. Turner (Eds.), *Handbook of the sociology of emotions* (pp. 87–113). New York, NY: Springer.
- Kopper, B. A., & Epperson, D. L. (1991). Women and anger: sex and sex-role comparisons in the expression of anger. *Psychology of Women Quarterly*, 15, 7–14.
- Kring, A. M. (2000). Gender and anger. In A. H. Fischer (Ed.), *Gender and emotion: social psychological perspectives* (pp. 211–231). Cambridge, UK: Cambridge University Press.
- Lewis, K. M. (2000). When leaders display emotion: how followers respond to negative emotional expression of male and female leaders. *Journal of Organizational Behavior*, 21, 221–234.
- Mattingly, M. J., & Sayer, L. C. (2006). Under pressure: gender differences in the relationship between free time and feeling rushed. *Journal of Marriage and the Family*, 68, 205–221.
- Mayne, T. J., & Ambrose, T. K. (1999). Research review on anger in psychotherapy. *JCLP/In Session: Psychotherapy in Practice*, 55, 353–363.
- McLeod, J. D., & Nonnemaker, J. M. (1999). Social stratification and inequality. In C. S. Aneshensel & J. C. Phelan (Eds.), *Handbook of the sociology of mental health* (pp. 321–344). New York: Kluwer Academic/Plenum.
- Mennino, S. F., Rubin, B. A., & Brayfield, A. (2005). Home-to-job and job-to-home spillover: the impact of company policies and workplace culture. *The Sociological Quarterly*, 46, 107–135.
- Milovchevich, D., Howells, K., Drew, N., & Day, A. (2001). Sex and gender role differences in anger: an Australian community study. *Personality and Individual Differences*, 31, 117–127.
- Mirowsky, J., & Ross, C. E. (1992). Age and depression. *Journal of Health and Social Behavior*, 33, 187–205.
- Mirowsky, J., & Ross, C. E. (1995). Sex differences in distress: real or artifact? *American Sociological Review*, 60, 449–468.
- Mirowsky, J., & Ross, C. E. (2003a). *Social causes of psychological distress* (2nd ed.). Hawthorne, New York: Aldine De Gruyter.
- Mirowsky, J., & Ross, C. E. (2003b). *Education, social status, and health*. Hawthorne, New York: Aldine De Gruyter.
- Pearlin, L. I. (1999). The stress process revisited: reflections on concepts and their interrelationships. In C. S. Aneshensel & J. C. Phelan (Eds.), *Handbook of the sociology of mental health* (pp. 105–123). New York: Kluwer Academic/Plenum.
- Pearlin, L. I., & Skaff, M. M. (1996). Stress and the life course: a paradigmatic alliance. *The Gerontologist*, 36, 239–247.
- Pearlin, L. I., & Turner, H. A. (1987). The family as a context of the stress process. In S. V. Kasl & C. L. Cooper (Eds.), *Stress and health: issues in research methodology* (pp. 143–165). New York: John Wiley and Sons.
- Ross, C. E., & Van Willigen, M. (1996). Gender, parenthood, and anger. *Journal of Marriage and the Family*, 8, 572–584.
- Ross, C. E., & Van Willigen, M. (1997). *Education and the subjective quality of life*. *Journal of Health and Social Behavior*, 38, 275–297.
- Russell, J. A., & Fehr, B. (1994). Fuzzy concepts in a fuzzy hierarchy: varieties of anger. *Journal of Personality and Social Psychology*, 67, 186–205.

- Sarah, A., Redston, P., & Campbell, A. (2003). Sex differences in social representations of aggression: Men justify, women excuse? *Aggressive Behavior*, 29(2), 128–133.
- Scherer, K. R., & Tannenbaum, P. H. (1986). Emotional experiences in everyday life: a survey approach. *Motivation and Emotion*, 10, 295–314.
- Scherer, K. R., & Wallbott, H. G. (1994). Evidence for universality and cultural variation of differential emotion response patterning. *Journal of Personality and Social Psychology*, 66, 310–328.
- Scherer, K. R., Wallbott, H. G., & Summerfield, A. B. (1986). *Experiencing emotion: a cross-cultural study*. Cambridge: Cambridge University Press.
- Schieman, S. (1999). Age and anger. *Journal of Health and Social Behavior*, 40, 273–289.
- Schieman, S. (2000). Education and the activation, course, and management of anger. *Journal of Health and Social Behavior*, 41, 20–39.
- Schieman, S. (2006). Anger. In J. E. Stets & J. H. Turner (Eds.), *Handbook of the sociology of emotions* (pp. 493–515). New York: Springer.
- Schieman, S., & Meersman, S. C. (2004). Neighborhood problems and health among older adults: received and donated social support and the sense of mastery as effect modifiers. *Journals of Gerontology: Social Sciences*, 59B, S89–S97.
- Schieman, S., Scott & Reid, S. (2008). Job authority and interpersonal conflict in the workplace. *Work and Occupations*, 35, 296–326.
- Schieman, S., Van Gundy, K., & Taylor, J. (2001). Status, role, and resource explanations for age differences in psychological distress. *Journal of Health and Social Behavior*, 42, 80–96.
- Sharkin, B. S. (1993). Anger and gender: theory, research, and implications. *Journal of Counseling and Development*, 71, 386–389.
- Shaver, P., Schwartz, J., Kirson, D., & O'Connor, C. (1987). Emotion knowledge: further exploration of a prototype approach. *Journal of Personality and Social Psychology*, 52, 1061–1086.
- Simon, R., & Nath, L. E. (2004). Gender and emotion in the United States: do men and women differ in self-reports of feelings and expressive behaviors? *American Journal of Sociology*, 109, 1137–1176.
- Smith, C. A., & Kirby, L. D. (2004). Appraisal as a pervasive determinant of anger. *Emotion*, 4, 133–138.
- Smith-Lovin, L. (1995). The sociology of affect and emotion. In K. S. Cook, G. A. Fine, & J. S. House (Eds.), *Sociological perspectives on social psychology* (pp. 118–148). Boston, MA: Allyn and Bacon.
- Stets, J. E., & Tsushima, T. M. (2001). Negative emotion and coping responses within identity control theory. *Social Psychology Quarterly*, 64, 283–295.
- Strachan, C. E., & Dutton, D. G. (1992). The role of power and gender in anger responses to sexual jealousy. *Journal of Applied Social Psychology*, 22, 1721–1740.
- Tavris, C. (1989). *Anger: the misunderstood emotion*. New York: Simon and Schuster.
- Thoits, P. A. (1989). The sociology of emotion. *Annual Review of Sociology*, 15, 317–342.

Chapter 20

Anger in the Context of Gender

Agneta H. Fischer and Catharine Evers

Abstract The simple picture of the angry male and the friendly female may be appealing, but it is oversimplified. Anger is an emotion that is experienced equally frequently by men and women because of goals that are blocked and other persons that transgress social rules. However, gender role practices and expectancies do affect the way in which men and women regulate their anger in different social contexts. Both social relations and social norms may make gender-specific features, expectancies, or roles salient, and it is these gender-specific features that influence the intensity, duration or quality of one's anger experience and expression, and the way in which one's anger is regulated. We especially analyze the impact of negative social appraisals of one's anger, which can be different for men and women, depending on the specific social context.

20.1 A contextual View on Gender and Emotions

There is a persistent stereotype in our society that women are more emotional than men. According to these commonly held beliefs, women are more emotionally responsive: They experience emotions more often and more intensely than men do and they also express them more overtly (Shields, 1991). Moreover, women seem to share and talk about their emotions more frequently, making them into a significant part of their life. If we unravel the concept of general emotionality into specific emotions, then emotions such as sadness, fear, or crying would be the stereotypical female emotions, implying powerlessness, whereas emotions such as contempt, annoyance, or anger would be the stereotypical examples of male emotions, indicating agency and power (Fischer, 1993). Focusing on anger, the stereotype thus suggests that men would more often experience and display anger than women. There are various reasons, however, to question this assumption.

First of all, the assignment of anger to men may suggest that anger is an innate and automatic reaction toward a specific event that is shaped foremost by biological and hormonal factors. Many studies on anger have shown that this is an incorrect representation of anger instances. Anger is an emotion that can be evoked in reaction to many different types of events, like being unable to get promotion whereas your colleague does (frustration, goal blockage), a friend not showing up at an appointment (rejection), a partner not taking you seriously (neglect), and someone gossiping about

A.H. Fischer (✉)

Department of Social Psychology, University of Amsterdam, 1018 WB, Amsterdam, The Netherlands
e-mail: a.h.fischer@uva.nl

you (criticism, social exclusion). Whether or not an individual reacts with anger therefore depends on how one construes the situation and on how one relates with the target of one's anger. In other words, one is not predisposed to react with anger toward a specific class of events, but one's anger depends on a variety of individual and contextual factors.

Second, anger is not a uniform experience or behavior. Anger may blend with other emotions (fear, shame, envy, disappointment, frustration, etc.), may consist of various appraisals and action tendencies, and may have different outlets (attacking, ignoring, criticizing, sneering, walking away, etc.), all resulting in another form of anger. However, there are shared and prototypical (although not necessary, see Kuppens, van Mechelen, Smits, & De Boeck, 2003) elements in most experiences that we call anger (but see Chapter 10 for a different position on this issue), such as appraising the situation as being caused by another person (other-blame), the tendency to change or act against the other person (tendency of antagonism), and the wish to force change upon someone (coercion, e.g., Fischer & Roseman, *in press*). How precisely anger is experienced and expressed depends on one's own role and position within a social context. These social factors mold our experiences and lead to the regulation of our anger, both in the short and in the long term. Whether one feels one's anger is justified, whether one thinks the other is really to blame, whether one believes the anger will help to change the other person, whether one thinks one's anger will backfire at oneself are all factors that determine whether one feels anger at all (or mere disappointment) and how one expresses it.

In this chapter we will develop the argument that there is no male or female anger. This does not mean that gender is irrelevant, but it means that there is no fixed pattern that accurately describes dispositional differences in anger for males or for females. We should leave behind the simple analogy of the Women-from-Venus and Men-from-Mars type; they are appealing, but they are oversimplified. Anger should be understood from the gender-specific features of the social context (see also Shields, 2002). Gender can affect one's anger, but only in interaction with the expectancies and appraisals of the social context. Social context on the one hand refers to the nature of the social relationship between oneself and others, which may be characterized by specific goals in the interaction, but also by unequal power or status relations, by love, or by respect for each other. Second, it refers to the social norms regarding one's anger and anger expression and the way in which the anger should be inhibited, reinforced, or changed. In some situations anger is inappropriate for women, but in other situations it is inappropriate for men. Both social relations and social norms may make gender-specific features, expectancies or roles salient and it is these gender-specific features that influence the intensity, duration or quality of one's anger experience and expression, and the way in which one's anger is regulated.

In this chapter we review empirical evidence with respect to gender differences in anger and anger expressions on the basis of a contextual framework, in which we combine Social Role Theory (Eagly, 1987, 1997; Eagly & Wood, 1999; Eagly, Wood, & Diekmann, 2000) and Social Appraisal Theory (Manstead & Fischer, 2001; Evers, Fischer, Rodriguez Mosquera, & Manstead, 2005). Both theories emphasize the general argument that our (emotional) behaviors are affected by the dynamics of the social relations in a particular context and may thus be helpful to explain some of the seeming inconsistencies in the empirical evidence on the relation between anger and gender. We will first review the empirical evidence on gender differences in anger and angry expressions, in order to demonstrate that these differences are strongly context dependent.

20.2 Evidence for the Angry Male or the Non-angry Female?

In contrast with the concept of gender-stereotyped anger, many studies have reported an absence of sex differences in the subjective experience of anger (see, e.g., Krings, 2000 for an overview). For

example, studies comparing men and women on trait anger (Deffenbacher et al., 1996; Kopper, 1991; Kopper & Epperson, 1991, 1996) have not found any sex differences in the likelihood to experience anger across a variety of situations. Moreover, most studies, whether diary studies (Oatley, 1998), autobiographical studies (Fischer & Roseman, in press) or other types of studies in which self-reported *intensity* of anger was measured, did not reveal any sex difference (Allen & Haccoun, 1976; Averill, 1983; Harris, 1994; Kring & Gordon, 1998; Wagner, Buck, & Winterbotham, 1993). This is supported by a meta-analysis on everyday occurrences of aggression, in which measures of anger were included, and in which no significant difference between men and women was found (Archer, 2004).

Moreover, if differences are found, as is the case in some studies, these do not support the stereotype, as women report more rather than less anger than do men (Brody, Lovas, & Hay, 1995; Fischer, Rodriguez-Mosquera, Van Vianen, & Manstead, 2004; Fischer & Roseman, 2007; Strachan & Dutton, 1992). Interestingly, the more intense anger on the part of women seems to be more prevalent in reaction to men than to women (Brody et al., 1995; Harris, 1994), when the anger is reported in intimate settings following condescending behavior by men (Buss, 1989; Frodi, 1977; Harris, 1994), or in Western cultures (Fischer et al., 2004; Strachan & Dutton, 1992). We will return to these contextual factors in more detail later in this chapter.

Studies examining the *expression* of anger also do not consistently support the stereotype of anger as a typically male emotion.¹ Studies using self-reports on the frequency of anger expressions (e.g., Allen & Haccoun, 1976; Balswick & Avertt, 1977; Campbell & Muncer, 1987; King & Emmons, 1990; Kopper & Epperson, 1991) failed to find sex difference on these measures, even when differences were found with respect to the expression of other emotions (e.g., women generally reporting more frequent expression of fear, sadness, happiness, or love). Moreover, using the distinction made by Spielberger et al. (1985) on anger-in and anger-out, the stereotype would lead us to believe that men would score higher on the anger-out scale, whereas women would score higher on anger-in. However, this expected difference in self-reported anger has not been found in normal populations (see Kring, 2000). Studies using scenarios on the other hand (e.g., Dosser, Balswick, & Halverson, 1983; Gross & John, 1995; Timmers, Fischer, & Manstead, 1998; Fischer & Roseman, 2007) have shown some sex differences, however, again opposite from what one would expect on the basis of the stereotype: if differences were found, women reported to express anger more often than men. This is especially the case when male partners were the targets of women's anger, which confirms that contextual determinants seem most important in analyzing gender differences.

Sex differences seem larger when considering *the way in which* anger is expressed, rather than the frequency of anger expression versus suppression, especially when we distinguish more overt, antagonistic, or powerful expressions of anger versus more indirect, covert, or powerless expressions. For example, some self-report studies have found that men more often physically aggress and verbally assault others than do women (Deffenbacher, Oetting, Lynch, & Morris, 1996), whereas women more often report to cry when angry compared to men (Eagly & Steffen, 1986; Frost & Averill, 1982; Lombardo, Cretser, Lombardo, & Mathis, 1983; Timmers et al., 1998). Differences in the nature of the anger expression are also found in meta-analyses on different types of aggression. Early meta-analyses on aggression have concluded that men engage more in physical aggression, but only slightly more in verbal aggression (Hyde, 1984; Eagly & Steffen, 1986). Some more recent meta-analytic reviews of aggression in real-world settings (Archer, 2004; Bettencourt & Miller, 1996) also

¹ We have included studies on aggression in this review of sex differences in anger expression, although the relationship with anger in these studies is not always explicit. We assume, however, that in the majority of these studies participants are angry to some extent.

report more physical aggression and somewhat less, but still more verbal aggression on the part of men, whether based on self-reports, peer reports, or teacher's reports.

When focusing on indirect aggression, such as gossiping, ignoring, or stonewalling, however, the direction of the sex difference reverts: Women show more indirect aggression than do men (Archer, 2004; Fischer & Roseman, 2007; Hess & Hagen, 2006). This more frequent use of indirect aggression that is already present in girls (Archer & Coyne, 2005; Xie, Carins, & Carins, 2002, but see Peets & Kikas, 2006 for an opposite finding) is explained by the larger social networks of women. Manipulating your friendships, for example by social exclusion, seems an excellent tool to force someone to apologize, or do what you want, without direct confrontation. Another explanation for the larger prevalence of indirect aggression by women is that they may be more sensitive to the potential negative implications of direct anger expressions (i.e., verbal or physical aggression), resulting in more indirect ways of showing your anger. We will return to this issue later in this chapter.

The conclusion that men use physical and verbal direct aggression more often, while women more often use indirect or social aggression should also be modified when taking contextual factors into account, however. In another meta-analysis of aggression in heterosexual relations, Archer (2000) has shown that women more frequently use physical aggression against their partners than men, although they are not more likely to inflict an actual injury on their partner. In other words, they aggress slightly more often toward their partners, but their aggression has less severe effects than that of their male counterparts. Interestingly, no systematic sex difference with regard to anger was found, which suggests that these sex differences in aggression cannot be accounted for by sex differences in anger.

Taken together, these studies do not support the existence of gender-stereotyped anger. First of all, sex differences in the subjective experience of anger are generally absent, and if they are found they are in contrast with the stereotype. Second, men often use more physical and verbal aggression than women, but this is not the case in intimate contexts where women are more angry and also express it in a direct way. Third, in some situations women express their anger differently, for example in powerless (e.g., crying) or indirect (e.g., gossiping) ways. These findings suggest that contextual factors affect the size and direction of the sex differences in anger and anger expression.

The fact that the stereotype is not confirmed does not necessarily mean that there are no differences between men and women in the way they experience and handle their anger. The point is rather that this relation is not simple and straightforward, but depends on the social context, which varies across studies. In some studies anger is measured in socially isolated circumstances, for example when using films or slides to evoke anger. Other studies investigated self-reported anger in imagined or hypothetical situations, such as autobiographical and vignette studies. Still other studies used real situations in which other people are involved. Moreover, the interpersonal contexts of these situations differ widely and include situations with close others (e.g., Fehr, Baldwin, Collins, Patterson, & Benditt, 1999), strangers (e.g., Blier & Blier-Wilson, 1989), couples having a conflict related to sexual jealousy (e.g., Strachan & Dutton, 1992), male vs. female targets of anger (e.g., Brody, 1993; Eagly & Steffen, 1986), anonymous vs. non-anonymous targets of anger (e.g., Evers et al., 2005; Lightdale & Prentice, 1994), and the target of anger being different from or the same as the object of anger (e.g., Timmers et al., 1998). Such features of the social context may appear to be crucial as they may render gender-specific expectancies and traits more or less salient.

This brings us to the question of how to explain the relation between sex, anger, and anger expressions. We propose a theoretical framework that combines Social Role Theory (Eagly, 1987; Eagly & Steffen, 1986) and Social Appraisal Theory (Evers et al., 2005; Manstead & Fischer, 2001). The core argument is that sex differences in anger are based on differences in the way the social context is construed. *Social Role Theory* provides a general framework by arguing that men and women

occupy different social roles and that these roles generate expectancies about gendered characteristics (stereotypically masculine and feminine traits and behaviors), such as helping, crying, or aggression. Whether or not men and women actually display these traits and behaviors depends on the salience of role-related expectancies in a specific social context and individual differences in these traits. We expect that role-specific concerns affect what makes one angry in the first place, but also whether one believes that one's anger is justified or not, and which anger expression would be most appropriate in which contexts. *Social Appraisal Theory* explains how one's emotional reaction to a situation is partly shaped by one's expectancies and subsequent appraisals of others' behavior in the same situation. Although both theories can explain gender differences in anger and anger expressions, they focus on different facets of the process. Social Role Theory emphasizes the normative role-related aspects of anger, whereas Social Appraisal Theory focuses on the more direct impact of others' reactions.

20.3 Anger and Role-Related Concerns

Social Role Theory (Eagly, 1987) emphasizes the different social roles in which both sexes are socialized. These roles are originally based on the distinct physical capacities of men and women, like bearing and feeding children versus protecting the family against an enemy, and have evolved in roles that are related to these capacities. Because women bear and nurture children, they are more likely than men to engage in domestic roles of primary caretaker of children, whereas men are more likely than women to occupy roles in the paid economy and to be primary family provider. These different roles do not only direct social behaviors that are required for those roles but also evoke stereotypic expectancies about the personal attributes associated with these roles, such as dominance and assertiveness versus caring and warmth. This division of social roles has pervaded all industrialized societies as a natural given, and therefore the stereotype that women are warm, nurturing, and emotional, whereas men are strong, dominant, and rational is widely shared.

Applying Social Role Theory to sex differences in the experience and expression of anger may provide an explanation of why anger may be differently evoked in men and women. The reason why one gets angry in the first place is because one's concerns are at stake (Frijda, 1986). These concerns reflect what one generally finds important in life, or more specifically at a particular moment, for example, one's goal to succeed for an exam, one's ambition to become a doctor, or one's desire to be as close as possible to one's lover. The engagement in a social role obviously determines one's major concerns, and therefore women who have the role of primary caretaker should be more concerned with the care of their children and the harmony in the relationship with their partner and children, whereas men in the provider role should be most concerned with job-related achievements. Anger arises when one's goals are blocked and when this negative outcome is seen as the result of an act of another person who could have avoided this (other-blame).

This differential distribution of men and women into social roles may explain why women are more often angry at and even show more direct anger toward their male partners than men toward their female partners. First of all, relationship concerns may be more important for women as part of their caretaker role and therefore may form the most frequent source of their anger toward their partners. Women may have strong expectancies about what their partners should do, feel, or say in order to show their love and commitment, and when these expectancies are not met, anger is easily evoked. The results from various self-report studies (Frodi, 1977; Harris, 1994) seem to support the conclusion that women are more concerned with relationship issues and therefore may be especially angry

when their partners neglect them or make condescending remarks toward them. The meta-analysis of anger in reaction to provocations (Bettencourt & Miller, 1996) also suggests that relationship concerns may be less important for men, who find insults that comprise achievement-related themes, such as intelligence, more infuriating. Indeed, sex differences were largest in the direction of men when receiving negative feedback about their intelligence.

Second, especially in more egalitarian relationships, men's refusal or neglect of household or care-taking tasks may be an additional reason to become angry at male partners. This anger may result in overt expressions, if the relationship is more egalitarian and when women perceive themselves as relatively powerful in this relationship. This is illustrated in an analysis of gender and culture differences in a large cross-cultural dataset on emotions, including anger (Fischer et al., 2004). Subjects were asked, among other things, to write down an instance of autobiographical anger and to report the intensity of this emotion. Although these data did not show significant sex or country differences for the intensity of anger in the overall sample, there was a significant interaction for the direct and overt expression of anger, labeled as antagonism, between sex of participant and the Gender Empowerment Measure (GEM). This is an index developed by the United Nations Development Program tapping the active participation of women in economic and political life in a specific country. In low GEM countries (such as Mexico, Brazil, India, or Zambia), women reported significantly less antagonism compared to men, whereas in high GEM countries (such as the Scandinavian and Western-European countries and the US), no differences between men and women were found. Moreover, a content analysis of the anger incidents described by the subjects revealed that women's antagonism in high GEM countries was more often caused by problems or conflicts in their intimate relations with men than that of women in low GEM countries.

The different distribution of men and women in more traditional or egalitarian roles may also give rise to different display rules that might result in different anger regulation processes. Display rules are learned early in life and prescribe when to show or mask one's anger. Men and women learn different display rules and social norms for the expression of anger (see Brody, 1985 for a review). According to traditional sex roles, girls are socialized to suppress hostile or antagonistic emotions, whereas boys are socialized to express their anger more freely (Brody, 1985). Extending this difference to adults, we would indeed expect the traditional caretaker role to inhibit overt anger expressions because these would endanger the quality of and harmony within the relationship, whereas the traditional provider role would underline the instrumental function of anger displays in certain contexts (see also Fischer & Roseman, 2007). Thus, overt or direct anger seems a more obvious part of the provider role, displaying assertiveness, confidence, and power, whereas covert or indirect anger would fit better with the female nurturing role. This is in line with the finding that women in traditional relations show less direct anger than women in egalitarian relations (Fischer, Rotteveel, Evers, & Manstead, 2004). Because the intensity of their anger does not differ, this difference in expression can be largely explained by the working of display rules. Thus, social norms that accompany gender roles direct the evaluation of one's own or others' emotions and subsequently lead to the regulation of one's anger.

However, the fact that women appear to become most angry and also express their anger most directly in intimate relations cannot be entirely explained on the basis of their traditional or egalitarian role concerns. Moreover, we may assume that women are more concerned with the harmony and quality of their intimate relation than with the quality of any other relation, so how can we explain their more frequent anger and anger expression? We assume that this is related with different perceptions of the effects or implications of men's and women's anger in different relationship contexts. Social Appraisal Theory more specifically conceptualizes how such appraisals of other persons' behavior in emotional situations may affect one's own emotional reaction.

20.4 Social Appraisal Theory and Anger Regulation

Social Appraisal Theory assumes that emotional reactions are determined not only by one's appraisal of the specific emotional event but also by the appraisal of the social context in which this event takes place. Individuals appraise others' emotional reactions, or the expected effects of one's emotions on others, and these so-called social appraisals influence their own emotions in different ways.

Two types of social appraisal may be distinguished (Manstead & Fischer, 2001). On the one hand, social appraisals should play a significant role in shaping the *experience* of an emotion, i.e., the way in which we evaluate an emotional event can be affected by our appraisal of how others (apparently) evaluate that same event (see Parkinson, 2001; Fischer et al., 2004). This type of social appraisal would especially operate in conditions where the emotional stimulus is ambiguous or low in intensity. In such situations others' appraisals are more diagnostic and more likely to shape one's own feelings. For example, when you are almost hit by a car when walking on the sidewalk, another person's enraged reaction toward the irresponsible behavior of the driver may make you feel very angry as well. In such cases social appraisals may be the underlying process of anger contagion. Second, social appraisals can also play a significant role in the *expression* of emotion, i.e., the ways in which people express their emotions are influenced by the expected social implications of these expressions. Imagining the negative effect of your anger with respect to your friendship with the other person, for example, may inhibit overt anger expressions, whereas considering the positive effects of one's anger may reinforce the intensity and directness of an anger expression. In sum, social appraisals refer to the appraisal of others' reactions on an emotional event, including others' reaction to one's own emotional reaction.

Social appraisals are thus by definition highly sensitive to variations in social context, but also by an individual's history of anger incidents. We argue that they may play an important role in explaining different emotional reactions by men and women. First of all, men and women may be differently sensitive to other persons' reactions toward their anger because of the aforementioned gender-specific role concerns. Men for example may be less concerned with how others evaluate their anger because anger does not negatively affect their masculine identity. Second, the expected social implications may be different for men and women depending on the social context. If one expects negative social implications of one's anger expression, it seems likely that one suppresses the anger, whereas the absence of such negative social appraisals may result in the overt expression of one's anger.

Various lines of evidence suggest that men and women indeed expect different social implications of their anger expressions and that negative social consequences of anger are more salient for women than for men. For example, various meta-analyses on sex differences in aggression have shown that perceptions of danger for retaliation (Bettencourt & Miller, 1996), perceived harm to the target of one's anger, or the anxiety about the possible negative consequences for others (Eagly & Steffen, 1986) are important predictors of sex differences in aggression. This is also apparent from a study involving autobiographical incidents of anger (Fischer & Roseman, 2007) that showed that women reported more verbal aggression than did men, but also more reconciliation. This may indicate that women more quickly feel regret about the negative effects of their anger and therefore try to make up. Moreover, women may also be more sensitive to withdrawal of positive social feedback (e.g., Stoppard & Gruchy, 1993). Women may also be more likely to empathize with the victim (Frodi, Macaulay, & Thome, 1977), and such an empathic motive may result in a greater tendency by women to suppress their anger (Timmers et al., 1998). It is unclear, however, whether this is mainly the case with partners, friends, and peers, or also with subordinates, or with strangers.

Men tend to anticipate these negative reactions about their own anger or aggression to a lesser extent and may in some situations even expect positive outcomes of their anger expression, such as

admiration or control. Indeed, studies on men's and women's representations of aggression suggest that women experience aggression as a loss of self-control, whereas men on the other hand experience it as a means of controlling others (Campbell & Muncer, 1987; Driscoll, Zinkivskay, Evans, & Campbell, 2006).

We tested the assumption that men and women expect different social implications of their intended anger reactions in a series of studies (Evers et al., 2005; Evers, Fischer, Manstead, & Rodriguez Mosquera, 2009). Participants were first asked to recall an autobiographical event in which they had experienced and expressed their anger (express condition) or a situation in which they had experienced, but suppressed their anger (suppress condition). Next, we asked various questions about the incident. As hypothesized, men and women differed in their social appraisals: When anger had been suppressed, women reported that they had anticipated more negative implications than men, and when anger was expressed, women reported fewer negative implications than men. This pattern of results suggests that women are more sensitive to the negative social consequences of their anger, resulting in its stronger regulation. Interestingly, the results also showed that women had a more intimate relationship with their provoker than men, but only in the express condition. This may suggest that women expect fewer negative implications of their anger, but especially if expressed toward their partner.

We therefore manipulated intimacy in a second scenario study. Because a pilot study showed that many participants mentioned a no-show at an appointment as the reason why they would become angry with both intimates and non-intimates, participants were asked to read a vignette describing a situation in which they are waiting in a restaurant for a person who is not there at the agreed time. Then a text message arrives on their phone, saying that the other is not going to show up because he or she is out with other people. Immediately after this message, they try to call the other person, but the phone is not answered. Participants had to imagine that the provoker in this scenario was either a partner or a colleague. The results showed that women indeed reported to express their anger more directly than men in the intimate condition. However, this sex difference in direct or overt anger expression was not related to sex differences in negative social appraisal. We assumed this was due to the fact that negative expectations are more closely associated with the suppression of anger and thus should be more salient when suppressing one's overt and direct anger, resulting in more indirect anger expressions, such as powerless expressions (e.g., disclosing your anger about the provoker to someone else or crying). This was confirmed in another scenario study, in which powerless expressions were included. Women expected more negative social implications of their initial anger than men and reported to express their anger in a more powerless way than men did. Negative social appraisals explained this sex difference in powerless anger expressions. In a final scenario study with a different social situation, this effect was exactly replicated.

Together these studies support one of the assumptions of Social Appraisal Theory that the anticipated effect of one's own emotional behavior on others is important for the regulation of anger expressions. Because we found stronger negative social appraisals for women and stronger effects of these appraisals, we may suggest on the basis of these results that women are more focused than men on the negative social implications of their anger expressions. This difference in social appraisal is responsible for at least some of the sex differences in anger expressions.

Because these studies are based on self-reports, which have the danger of hindsight interpretations by subjects, we also conducted an experiment in which anger was evoked in vivo in the laboratory, in different social contexts. In this experiment (Evers et al., 2005) participants were made angry by very demeaning feedback about their writing from a bogus (nonexistent) fellow student. As a consequence of this false negative feedback, participants did not receive a financial reward. Moreover, the feedback also contained a note indicating that participants were naïve and had an immature point of view. Participants were then randomly assigned to either a *social* condition, in which they

expected to meet the fellow student who had provided the negative feedback, or to a *non-social* condition, in which they had no such expectation. Subsequently, participants were instructed to allocate hot sauce to the fellow student, which he or she had to taste in an ostensibly unrelated study. This “hot sauce paradigm” is a method that has proven effective as a way of implicitly measuring anger-related actions (e.g., Bushman, Baumeister, & Philips, 2001; Lieberman, Solomon, Greenberg, & McGregor, 1999).

Results indicated that men and women experienced anger equally intense; however, they differed in their anger expression. Women expressed anger to a lesser extent than men, but only in the social condition. In other words, when women expected to meet the fellow student, they allocated less hot sauce than men. Moreover, women again focused more strongly on the negative social implications of their anger. These negative social appraisals partly accounted for the sex difference in anger expression.

Together the findings of these studies on social appraisal enhance our understanding of how social processes affect anger and the expression of anger. Men and women are both sensitive to others’ reactions, yet women seem to be especially sensitive to the negative implications of their anger displays, resulting in either more indirect anger expressions or more reconciliation after direct expressions.

20.5 Conclusion

In this chapter we have argued that there is no such phenomenon as the angry male and the non-angry or kind and relaxed female. There is ample evidence that men and women become angry equally often, and equally intense, although there may be gender differences depending on what makes one angry. The most striking difference in this respect seems that women are more often angry at their male partners than vice versa, especially in more egalitarian relationships. This supports the idea that role-related concerns influence not only whether one blames another person but also whether one feels that one’s anger is justified.

When considering the expression of anger, we may conclude that gender differences are most apparent in the way in which one’s anger is expressed. The variety of findings relating to men’s and women’s anger expressions suggests more ambivalence on the part of women toward their own anger. Women seem to have a preference for less antagonistic and more indirect expressions, albeit not toward their partners. These indirect anger displays may be the result of negative appraisals of others’ reactions, for example, fear of retaliation or of loss of self-control or of hurting the other person. Thus, women may have similar motivational goals when angry as men have, but may more often than men be concerned about the negative effects of their anger. There are several explanations for this larger salience of negative social appraisals for women.

In general, women’s traditional role concerns do not fit with overt and direct anger expressions, as this may deteriorate the quality of the relationship and undermine the position of man. Although intimate relations and the accompanying role division between men and women can be characterized as more egalitarian nowadays, women still seem to be more attuned to their social networks. Several lines of research support these assumptions. Taylor and colleagues (2000), for example, propose that female behavioral stress responses are characterized by a *tend-and-befriend*. Tending involves activities to protect the self, and befriending is the creation and maintenance of social networks that may help in this process. For women, negative social appraisals and the resulting regulation of their anger may reflect the safeguarding of their social networks because direct anger could harm their relations. Further, Cross and colleagues (Cross, Gore, & Morris, 2003; Cross & Morris, 2007) have

shown that individuals with a highly relational self think and behave in ways that nurture important or close relationships. These individuals also take the implications of their own anger expression more strongly into account. Because women more often appear to have an interdependent self than men, this research confirms that women are more focused on the relational consequences of their anger because of the importance of close relationships.

This focus on relational concerns may also explain why women would express more anger in intimate relations. We have argued that more egalitarian roles may provide not only more reasons for women to become angry but also more justifications for their anger, especially when their partners do not meet expectations or agreements. In these contexts negative social appraisals may become less salient, whereas feelings that one's anger is just and may have the intended effect of changing the partner's behavior become more prominent, leading to more direct anger expressions.

In conclusion, anger is not an individual disposition present in men and absent in women, nor is it an automatic reaction to a certain class of events. Anger may vary from a simple reaction to goal blockage to a more complex reaction to an insult from an intimate and it is regulated in all its facets, even starting with whether it is elicited or not. Gender differences are especially apparent in the way in which anger is expressed and are at least partly determined by the anticipation of positive or negative effects of one's anger. These different expectations are related to role-related and relational concerns and result from the way in which this anger is construed and acted upon in the social context.

References

- Allen, J. G., & Haccoun, D. M. (1976). Sex differences in emotionality: A multi-dimensional approach. *Human Relations*, 29, 711–722.
- Archer, J. (2000). Sex differences in aggression between heterosexual partners: A meta-analytic review. *Psychological Bulletin*, 126, 651–680.
- Archer, J. (2004). Sex differences in aggression in real-world settings: A meta-analytic review. *Review of General Psychology*, 8, 291–322.
- Archer, J., & Coyne, S. M. (2005). An integrated review of indirect, relational, and social aggression. *Personality and Social Psychology Review*, 9, 212–230.
- Averill, J. R. (1983). Studies on anger and aggression: Implications for theories of emotion. *American Psychologist*, 38, 1145–1160.
- Balswick, J., & Avertt, C. (1977). Differences in expressiveness: Gender, interpersonal orientation, and perceived parental expressiveness as contributing factors. *Journal of Marriage and Family*, 39, 121–127.
- Bettencourt, B. A., & Miller, N. (1996). Gender differences in aggression as a function of provocation: A meta-analysis. *Psychological Bulletin*, 119, 422–447.
- Blier, M. J., & Blier-Wilson, L. A. (1989). Gender differences in self-rated emotional expressiveness. *Sex Roles*, 21, 287–295.
- Brody, L. R. (1985). Gender differences in emotional development: A review of theories and research. *Journal of Personality*, 53, 102–149.
- Brody, L. R. (1993). On understanding gender differences in the expression of emotion: Gender roles, socialization, and language. In S. Ablon, D. Brown, E. Khantzian, & J. Mack (Eds.), *Human feelings: Explorations in affect development and meaning* (pp. 87–121). New York: Analytic Press.
- Brody, L. R., Lovas, G. S., & Hay, D. H. (1995). Gender differences in anger and fear as a function of situational context. *Sex Roles*, 32, 47–78.
- Bushman, B. J., Baumeister, R. F., & Philips, C. P. (2001). Do people aggress to improve their mood? Catharsis beliefs, affect regulation opportunity, and aggressive responding. *Journal of Personality and Social Psychology*, 81, 17–32.
- Buss, D. M. (1989). Conflict between the sexes: Strategic interference and the evocation of anger and upset. *Journal of Personality and Social Psychology*, 5, 735–747.
- Campbell, A., & Muncer, S. (1987). Models of anger and aggression in the social talk of women and men. *Journal for the Theory of Social Behaviour*, 17, 489–511.

- Cross, S. E., Gore, J. S., & Morris, M. L. (2003). The relational-interdependent self-construal, self-concept consistency, and well-being. *Journal of Personality and Social Psychology*, 85, 933–944.
- Cross, S. E. & Morris, M. L. (2007). Getting to know you: The relational self-construal, relational cognition, and well-being. *Personality and Psychological Bulletin*, 29, 512–523.
- Deffenbacher, J. L., Oetting, E. R., Lynch, R. S., & Morris, C. A. (1996). The expression of anger and its consequences. *Behavior Research and Therapy*, 34, 575–590.
- Deffenbacher, J. L., Oetting, E. R., Thwaites, G. A., Lynch, R. S., Baker, D. A., Stark, R. S., et al. (1996). State-trait anger theory and the utility of the trait anger scale. *Journal of Counseling Psychology*, 43, 131–148.
- Dosser, D., Balswick, J., & Halverson, C. (1983). Situational context of emotional expressiveness. *Journal of Counseling Psychology*, 30, 375–387.
- Driscoll, H., Zinkivskay, A., Evans, K., & Campbell, A. (2006). Gender differences in social representations of aggression: The phenomenological experience of differences in inhibitory control? *British Journal of Psychology*, 97, 139–153.
- Eagly, A. H. (1987). *Sex differences in social behavior: A social-role interpretation*. Hillsdale, NJ: Erlbaum.
- Eagly, A. H. (1997). Sex differences in social behavior: Comparing social role theory and evolutionary psychology. *American Psychologist*, 52, 1380–1383.
- Eagly, A. H., & Steffen, V. J. (1986). Gender and aggressive behavior: A meta-analytic review of the social psychological literature. *Psychological Bulletin*, 100, 309–330.
- Eagly, A. H., & Wood, W. (1999). The origins of sex differences in human behavior. Evolved dispositions versus social roles. *American Psychologist*, 54, 408–423.
- Eagly, A. H., Wood, W., & Diekmann, A. B. (2000). Social role theory of sex differences and similarities: A current appraisal. In Eckes, T. & Trautner, H. M. (Eds.), *The developmental social psychology of gender* (pp. 123–174). Mahwah, NJ: Lawrence Erlbaum Associates Publishers.
- Evers, C., Fischer, A. H., Manstead, A. S. R., & Rodriguez Mosquera, P. M. (under review). *Sex differences in anger expression: The role of social appraisals*.
- Evers, C., Fischer, A. H., Rodriguez Mosquera, P. M., & Manstead, A. S. R. (2005). Anger and social appraisal: A “spicy” sex difference? *Emotion*, 5, 258–266.
- Fehr, B., Baldwin, M., Collins, L., Patterson, S., & Benditt, R. (1999). Anger in close relationships: An interpersonal script analysis. *Personality and Social Psychology Bulletin*, 25, 299–312.
- Fischer, A. H. (1993). Sex differences in emotionality: Fact or stereotype. *Feminism and Psychology*, 3, 303–318.
- Fischer, A. H., Rodriguez-Mosquera, P. M., Van Vianen, A. E. M., & Manstead, A. S. R. (2004). Gender and culture differences in emotion. *Emotion*, 4, 87–94.
- Fischer, A. H., & Roseman, I. J. (2007). Beat them or ban them: The characteristics and social functions of anger and contempt. *Journal of Personality and Social Psychology*, 93, 103–115.
- Fischer, A. H., Rottevel, M., Evers, C., & Manstead, A. S. R. (2004). Emotional assimilation: How we are influenced by others’ emotions. *Cahier de Psychologie Cognitive*, 22, 223–245.
- Frijda, N. H. (1986). *The Emotions*. Cambridge, UK: Cambridge University Press.
- Frodi, A. (1977). Sex differences in the perception of a provocation: A survey. *Perceptual and Motor Skills*, 44, 113–114.
- Frodi, A., Macaulay, J., & Thome, P. R. (1977). Are women always less aggressive than men? A review of the experimental literature. *Psychological Bulletin*, 84, 634–660.
- Frost, W. D., & Averill, J. R. (1982). Differences between men and women in the everyday experience of anger. In J. R. Averill (Eds.), *Anger and aggression: An essay on emotion* (pp. 281–317). New York: Springer-Verlag.
- Gross, J. J., & John, O. P. (1995). Facets of emotional expressivity: Three self-report factors and their correlates. *Personality and Individual Differences*, 19, 555–568.
- Harris, M. B. (1994). Gender of subject and target as mediators of aggression. *Journal of Applied Social Psychology*, 24, 453–471.
- Hess, N. H., & Hagen, E. H. (2006). Sex differences in indirect aggression: Psychological evidence from young adults. *Evolution and Human Behavior*, 27, 231–245.
- Hyde, J. S. (1984). How large are gender differences in aggression? A developmental meta-analysis. *Developmental Psychology*, 20, 722–736.
- King, L., & Emmons, R. A. (1990). Conflict over emotional expression: Psychological and physical correlates. *Journal of Personality and Social Psychology*, 58, 864–877.
- Kopper, B. A. (1991). Role of gender, sex role identity, and Type A behavior in anger expression and mental health functioning. *Journal of Counseling Psychology*, 40, 232–237.
- Kopper, B. A., & Epperson, D. L. (1991). Women and anger. Sex and sex role comparisons in the expression of anger. *Psychology of Women Quarterly*, 15, 7–14.

- Kopper, B. A., & Epperson, D. L. (1996). The experience and expression of anger: Relationships with gender, gender role socialization, depression, and mental health functioning. *Journal of Counseling Psychology*, 43, 158–165.
- Kring, A. M. (2000). Gender and anger. In A. H. Fischer (Ed.), *Gender and emotion: Social psychological perspectives* (pp. 211–231). Cambridge: Cambridge University Press.
- Kring, A. M., & Gordon, A. H. (1998). Sex differences in emotion: Expression, experience, and physiology. *Journal of Personality and Social Psychology*, 74, 686–703.
- Kuppens, P., van Mechelen, I., Smits, D. J. M., & De Boeck, P. (2003). The appraisal basis of anger: Specificity, necessity and sufficiency of components. *Emotion*, 3, 254–269.
- Lieberman, J. D., Solomon, S., Greenberg, J., & McGregor, H. A. (1999). A “hot” new way to measure aggression: Hot sauce allocation. *Aggressive Behavior*, 25, 331–348.
- Lightdale, J. R., & Prentice, D. A. (1994). Rethinking sex differences in aggression: Aggressive behavior in the absence of social roles. *Personality and Social Psychology Bulletin*, 20, 34–44.
- Lombardo, W. K., Cretser, G. A., Lombardo, B., & Mathis, S. L. (1983). Fer cryin’ out loud – There’s a sex difference. *Sex Roles*, 9, 987–995.
- Manstead, A. S.R., & Fischer, A. H. (2001). Social appraisal: the social world as object of and influence on appraisal processes. In K. R. Scherer, A. Schorr, & T. Johnstone (Eds.), *Appraisal processes in emotion: Theory, method, research* (pp. 221–232). Oxford: Oxford University Press.
- Oatley, K. (1998). State of the art: Emotion. *The Psychologist*, 11, 286–288.
- Parkinson, B. (2001). Putting appraisal in context. In K. R. Scherer, A. Schorr, & T. Johnstone (Eds.), *Appraisal processes in emotion: Theory, method, research* (pp. 173–186). New York: Oxford University Press.
- Peets, K., & Kikas, E. (2006). Aggressive strategies and victimization during adolescence: Grade and gender differences, and cross-informant agreement. *Aggressive Behavior*, 32, 68–79.
- Shields, S. A. (1991). Gender in the psychology of emotion: A selective research review. In K. T. Strongman (Ed.), *International review of studies on emotion* (pp. 227–245). New York: Wiley.
- Shields, A. (2002). *Speaking from the heart: Gender and the social meaning of emotion*. New York: Cambridge University press.
- Spielberger, C. D., Johnson, E., Russell, S., Crane, R., Jacobs, G., & Worden, T. (1985). The experience and expression of anger: Construction and validation of an anger experience scale. In M. A. Chesney & R. H. Rosenman (Eds.), *Anger and hostility in cardiovascular and behavioral disorders* (pp. 5–30). New York: McGraw Hill.
- Stoppard, J. M., & Gruchy, C. G. (1993). Gender, context, and expression of positive emotion. *Personality and Social Psychology Bulletin*, 19, 143–150.
- Strachan, C. E., & Dutton, D. G. (1992). The role of power and gender in anger responses to sexual jealousy. *Journal of Applied Social Psychology*, 22, 1721–1740.
- Taylor, S. E., Klein, L. C., Lewis, B. P., Gruenewald, T. L., Gurung, R. A.R., & Updegraff, J. A. (2000). Biobehavioral responses to stress in females: Tend-and-befriend, not fight-or-flight. *Psychological Review*, 107, 411–429.
- Timmers, M., Fischer, A. H., & Manstead, A. S.R. (1998). Gender differences in motives for regulating emotions. *Personality and Social Psychology Bulletin*, 9, 974–985.
- Wagner, H. L., Buck, R., & Winterbotham, M. (1993). Communication of specific emotions: Gender differences in sending accuracy and communication measures. *Journal of Nonverbal Behavior*, 17, 29–53.
- Xie, H., Carins, R. B., & Carins, B. D. (2002). The development of social aggression and physical aggression: A narrative analysis of interpersonal conflicts. *Aggressive Behavior*, 28, 341–355.

Chapter 21

Madmen: An Evolutionary Perspective on Anger and Men's Violent Responses to Transgression

Daniel M.T. Fessler

Abstract Though often described as leading to costly and irrational decisions, anger's effects on behavior are understandable when anger is viewed as an adaptation favored by natural selection. Anger motivates responses to transgression despite our propensity to discount the future, truncating ongoing transgressions and deterring additional transgressions. An evolutionary perspective sheds light on differences in anger's effects on male and female behavior. Due to differences in the variance of reproductive success between men and women, men can be viewed as playing a higher stakes game than women, one in which the fitness consequences of transgression are generally greater. Selection has therefore favored more risky aggressive responses to transgressions in men, with corresponding differences in the propensity to engage in other forms of risky behavior. This explains both robust sex differences in rates of violence and parallel patterns in other forms of risk taking. Similarly, the cost/benefit ratio of aggression and other forms of risk taking changes both across the lifecycle and as a function of reproductive status; involvement in violence and other risky behavior directly tracks such changes. Matching the physical architecture to the tasks at hand, changes in both male musculature and underlying neurophysiology likewise correspond to changes in the payoffs of aggressive responses to transgression.

Both ethnographic accounts (Lee, 1993; Burbank, 1994; Chagnon, 1997; Gladwin & Sarason, 1953; Johnson, Johnson, & Baksh, 1986; Myers, 1988) and Western judicial records (see Daly & Wilson, 1988; Ghiglieri, 1999) suggest that the emotion that English speakers label "anger" figures prominently in violent conflict. Although both the eliciting conditions and the local construals of anger vary cross-culturally, anger is likely one of the most universally identifiable emotions (Ekman, 1994; Haidt & Keltner, 1999; Johnson et al., 1986; Myers, 1988). Western observers frequently view anger as a destructive, or at least counterproductive, emotion. However, given the costs associated with this trait, the species-typical propensity to experience anger could only have evolved and been maintained if, in ancestral populations, possessing this attribute enhanced the probability of individual survival and reproduction. Note that this does not suppose that anger continues to have such effects today – due to changes in our social and ecological environments, features that increased survival and reproduction in the world of our ancestors can now have the opposite effect (witness, for example, the contemporary health consequences of our evolved preferences for sugar and fat). Note also

D.M.T. Fessler (✉)

Department of Anthropology and Center for Behavior, Evolution, and Culture, University of California,
Los Angeles, CA 90095-1553, USA
e-mail: dfessler@anthro.ucla.edu

that this position does not imply moral validation of anger or its consequences, as scientific explanations do not constitute ethical justifications. With these caveats in mind, if we are to understand anger's impact on our lives today, we must begin by asking what adaptive function it performed in the past.

The first step in inferring adaptive function is to consider the circumstances in which the phenomenon occurs. I begin with two observations: (a) anger is elicited by transgression against the actor or those whom the actor holds dear, and (b) the most common behavioral outcome of anger is an attempt to harm the transgressor. Transgression can be defined as the imposition of a cost on an actor that the actor does not pay willingly, where such unwillingness derives from the actor's valuation of his own welfare relative to the other party's welfare (see Tooby, Cosmides, & Price, 2006). For example, a doting mother awoken by her squalling infant does not construe the disturbance as a transgression, yet would likely do so had she been awoken by a neighbor's dog (see also Chapter 31 by Van Kleef and also Chapter 19 by Schieman, this volume).

Across species, access to resources, such as foodstuffs and mates, is a principal determinant of biological fitness. In nonhuman social species, such access is largely determined by dominance, the ability to displace a rival by dint of force or the threat thereof (Ellis, 1995). As the foundation of social hierarchies, in humans, dominance has been partially replaced by prestige, deference that is freely granted in recognition of merit (Henrich & Gil-White, 2001). However, while prestige is important in all societies, dominance continues to play a role in human hierarchies, and this is likely particularly true in small-scale societies, akin to those of our ancestors, that lack an organized state to effectively regulate violent conflicts. Correspondingly, in ancestral human populations, differences in the willingness and ability to truncate and deter transgressions will have been a determinant of differences in the ability to maintain control over resources, and hence of differences in fitness. This suggests that natural selection will have favored a disposition to respond aggressively to transgression. However, anger is much more complex than this conclusion implies. Consider the phrase "blinded by anger": As is equally true of Southern Californians, Bengkulu Malays (Fessler, 2001) and Pintupi Aborigines (Myers, 1988), people recognize that angry individuals are often aware only of their desire to inflict costs on those who have endangered them – other considerations either fade from consciousness or else lose their motivational salience (cf. "out of control" Chapter 22 by Potegal, this volume). This aspect of anger is almost certainly not accidental, but rather constitutes a key component of an evolved mechanism designed to limit transgression. To understand why, we must first consider a pervasive feature of the relationship between cost and time.

21.1 Time Discounting and the Response to Transgression: Why "Over-reacting" Pays Off

Humans, like other vertebrates, steeply discount the future – the motivational salience of rewards or punishments declines rapidly as the intervening delay increases (Frederick, Loewenstein, & O'Donoghue, 2002). The future plays a critical role in calculations concerning the utility of responding to transgression, since an aggressive response can benefit the responder not only by truncating the ongoing transgression but also by deterring future transgressions. Note, however, that even if the actual costs that multiple transgressions inflict on an individual are the same whether the transgression occurs in the present or in the future, because the future is steeply discounted, transgressions will possess wildly varying punitive salience as a function of time from the present – the longer the predicted delay before a potential transgression, the less motivational significance it holds for the victim. As a consequence, if the costs of responding to an ongoing transgression greatly exceed the benefits of truncating it, discounters may fail to respond due to subjective devaluation

of the additional benefit of deterring future transgressions. The result of such myopia, however, is that, over the long term, the individual will incur sizable costs – one can be “nickel-and-dimed to death” because one is never willing to pay the price of confronting the thief caught stealing a nickel. It is therefore advantageous to commit early to a strategy of significant deterrence. Emotions in general may motivate such commitment (Schelling, 1980), and anger in particular may usefully blind us to the immediate costs of responding to transgression, counteracting some of the detrimental consequences of time discounting (Hirshleifer, 1987; Frank, 1988).

If one crucial function of the response to a current transgression is the deterrence of future transgressions, then the responder should be willing to incur costs up to the sum of the costs of all possible future transgressions reduced by the probability that each will occur. In situations that hold the potential for a large number of future interactions, and hence a large number of possible transgressions, this sum may be huge. Accordingly, the costs that an individual ought to be willing to incur in responding to a given transgression may vastly exceed the costs that the given transgression inflicts on him – a strategically appropriate response will often appear “disproportionate” to the transgression. Such responses dramatically raise the costs inflicted on individual transgressors, with corresponding increases in the likelihood of truncation and deterrence. Anger is thus partly explicable as a mechanism produced by economically rational natural selection in order to operationalize this logic in a species of steep time discounters – when we are angry we are often motivated to inflict suffering on the transgressor that greatly exceeds our own, even at great expense to ourselves (Frank, 1988; McGuire & Troisi, 1990; Edwards, 1999, pp. 140–141).

The utility of responding disproportionately to transgression hinges on discouraging future transgressions, of which there are two types, namely (a) additional transgressions by the same transgressor, and (b) transgressions committed by others. The propensity to respond in a seemingly disproportionate fashion can evolve only when certain conditions are met. First, social relationships must extend beyond single interactions if type (a) transgressions are to be discouraged. Second, if type (b) transgressions are to be precluded, individuals must be able to (i) witness (as third parties) an actor’s responses to others’ transgressions and/or (ii) detect others’ willingness, or lack thereof, to transgress against a given actor. All of these criteria are fulfilled in many primate species, and the same was almost certainly true of our ancestors. Furthermore, the existence of language makes it possible for an actor to establish a *reputation*, that is, others can learn of his propensity to react disproportionately even before meeting him (Frank, 1988; Daly & Wilson, 1988; Schelling, 1980). Because a reputation can greatly enhance deterrence effects, the evolution of language presumably increased the (already strong) selective pressure favoring the presence of anger as a mechanism motivating aggressive response to transgressions.

21.2 Sex Differences in the Outcome Behaviors Associated with Anger

As reviewed by both Fischer and Evers and Schieman in this volume, contrary to many folk models, research suggests that men and women do not reliably differ in the frequency or intensity of their experience of anger (if anything, women may slightly exceed men in this regard). From an evolutionary perspective, this similarity across the sexes is understandable given that the overarching logic of the need to truncate and deter transgressions is the same for men and women. Where men and women do differ, however, is in the behaviors resulting from anger. Whereas men are more likely to approach the targets of their anger directly, and more likely to physically aggress against them, women are more likely to seek the aid of allies, and to engage in indirect aggression through the manipulation of social relationships and attempts to inflict reputational damage (see Chapter 20 by Fischer and Evers, this volume; Chapter 19 by Schieman, this volume; Campbell, 2002,

pp. 90–94; Kring, 2000). Although some authors try to downplay differences in anger-related outcome behaviors between men and women, the marked sex difference in participation in lethal violence suggests that, with regard to one of the most fitness-relevant forms of action, anger indeed leads to very different behaviors in the two sexes.

Around the globe, men have a near-monopoly on murderous violence (Daly & Wilson, 1988; Daly & Wilson, 1990; Ghiglieri, 1999). Critical to the present argument, this is true not only of instrumental violence but, more importantly, of violence linked to altercations – whereas it is arguable whether anger plays a role in the former, no such ambiguity surrounds the latter. Equally telling, despite the fact that simply apologizing or walking away often de-escalates altercations (Chapter 22 by Potegal, this volume), men are vastly more likely not only to kill but also to be killed in spontaneous acts of violence, a sex difference that, with few exceptions, holds across disparate cultures (Gartner, 1990; Daly & Wilson, 2001; Lambert, 1994).

Do sex differences in involvement in homicide truly reflect differences in the way in which anger influences men's and women's willingness to engage in potentially costly confrontations? Might it not simply be that males are more likely to be involved in violent confrontations because the costs of violence are usually lower for them than they are for women, with homicide representing an erroneous calculation on the victim's part? Two different avenues of research suggest that the answer is no.

First, while physical size is a partial determinant of sex differences in participation in violence (Felson, 1996), the relationship between coercive power and aggression is not as simple as this implies. Sell, Tooby, and Cosmides (2009) compared measures of physical strength in US subjects with self-reports of the frequency and intensity of anger, proneness to respond aggressively to transgressions, involvement in physical fights, and endorsement of coercive tactics; similar comparisons were also made in a second sample using anthropometric proxies for strength. In both studies, strength was positively correlated with all of the dependent variables, but only for men – in contrast to the prediction that follows from the claim that sex differences in violence are due to differences in size and strength, physically strong women are not more angry, more confrontational, more violent, or more coercive than are physically weak women. The relationship between strength, anger, and violence, it seems, is a uniquely male one.¹

A second line of evidence arguing against the claim that sex differences in participation in violence are due primarily to sex differences in size and strength derives from a more careful reading of the epidemiological data. If women's smaller size and lesser strength were the limiting factor in their involvement in violence, then we would expect female/female homicide rates to be equivalent to male/male homicide rates, yet this is not the case. Between 1976 and 1998 male/male killings accounted for 65% of all homicides committed in the United States, while female/female killings constituted only 2.4% (Fox & Zawitz, 2000). Across societies ranging in scale from modern nation-states to small hunter-gatherer bands akin to those in which our ancestors lived, men are vastly more likely to kill men than women are to kill women (Daly & Wilson, 2001). Moreover, size and strength are no longer unique determinants of the potential costs of involvement in anger-fueled altercations – in the contemporary United States there are no significant obstacles to female firearm ownership, yet, between 1976 and 1987, women committed only 13.3% of all homicides involving a gun (Kellermann & Mercy, 1992). Even if we consider the context in which women are most likely to kill with a gun, the sex difference does not disappear: Murder of a spouse or intimate acquaintance

¹ Congruent with Sell et al.'s findings, Klinesmith et al. (2006) demonstrated that handling a gun (which, like physical strength, is a determinant of the outcome of conflict) increases aggression in men; the above perspective suggests that any such effect should be reduced, or absent, in women.

constituted 54.2% of all firearm homicides committed by women in the United States (in contrast to only 11.1% of male firearm homicides) (Kellermann & Mercy, 1992). Spousal murder is an “equal opportunity” crime, since in many cases, a firearm used to kill a spouse was present in the home prior to the assault, i.e., roughly equivalent access existed for both husband and wife (Foster, Veale, & Fogel, 1989; Bailey et al., 1997; Kellermann, Somes, Rivara, Lee, & Banton, 1998). Nevertheless, in the United States women commit only 42.6% of firearm spousal homicides, and even this figure gives a vastly inflated impression of the female propensity toward spontaneous violence since, unlike husbands, wives often kill in self-defense (Kellermann & Mercy, 1992; Wilson & Daly, 1992b). In short, it is untenable to argue that the sex difference in the propensity for direct, potentially violent aggression following elicitation of anger is due to some role-related aspect of persons deriving from morphological, rather than psychological, differences between men and women. Indeed, rather than being the cause of behavioral differences between men and women, differences between male and female bodies likely derive from the same evolutionary factors as do the psychological differences in question.

21.3 The Evolutionary Origins of Sex Differences in Responses to Transgression

The higher the stakes in any transgression, the more willing both parties should be to expose themselves to risks in order to win out over the other. Undoubtedly, in ancestral human populations, there were times when women faced high-stakes transgressions – think, for example, of the dangers posed by an infanticidal enemy, a rapist, or a desperate competitor during a famine. However, in contrast to the episodic and irregular nature of these events, for men, high-stakes transgressions are likely to have been both more frequent and more inevitable. As has been extensively argued by Margo Wilson and Martin Daly (see Daly & Wilson, 2001 for an overview), the reason lies in the nature of human reproduction.

Ethnographic surveys of marriage patterns around the globe suggest that, at a societal level, the most common human reproductive pattern is one of mild polygyny, meaning that many men marry a single a wife, some men never marry, and a few men marry multiple wives (Murdock, 1967). Convergent evidence that this reproductive pattern characterized ancestral human populations derives from studies of the human body. Although there is variation across extant populations (Gaulin & Boster, 1992), men are typically 4–10% larger than women and considerably stronger. Comparisons across primates reveal that the degree of sexual dimorphism in size characteristic of a species in part corresponds with the degree of polygyny in the mating system (Plavcan, 2001). This variation can be understood in terms of the factors that determine the reproductive success of the two sexes. Due to the costs of gestation and lactation, mammalian females must invest a great deal in each offspring. The energetic and nutritional demands of this investment are such that access to resources is a principal determinant of female mammalian reproductive success. In contrast, although, as the human case demonstrates, males are capable of extensive investment in their offspring, this is not a prerequisite for male reproduction, and many primate males invest little or nothing in this regard. Hence, for males, reproductive success is principally determined by the degree of access to females. Given that sex ratios are generally balanced, polygyny thus necessarily introduces competition into male mating behavior – because access to females is the primary determinant of male reproductive success, the more polygynous the mating system, the higher the stakes for each male competitor, as the winners will leave many more offspring than the losers (who may not leave any at all). The greater the stakes in male–male competition, the more that it pays to invest in costly armaments. Hence, whereas female body size appears to reflect efficient exploitation of the resources available

in the given environment, in polygynous species, males appear to be “overbuilt” – their greater size and strength, inefficient when viewed in ecological terms, are presumably favored by natural selection in part due to the reproductive benefits that successful males achieve through dominance.

In seasonally breeding animals, male–male conflicts cluster in space and time around fertile females, as males attempt to both transgress against other males and defend against such transgressions in order to monopolize reproductive opportunities; outside of the breeding season, males are more tolerant of transgressions, and less interested in transgressing, since the stakes are much lower. However, in species such as ours that lack a distinct breeding season, sensitivity to transgressions cannot be regularly relaxed. Moreover, in long-lived, highly social creatures like ourselves, the scope of male–male interactions with potential reproductive consequences expands well beyond direct conflicts over access to females or territory: because even transgressions far removed from reproductive opportunities may set precedents that later encourage or discourage such direct-payoff transgressions, males must be vigilant in a wide variety of contexts. The existence of linguistically encoded reputations further increases this effect, enhancing the potential importance of a huge variety of transgressions. Lastly, the fact that, unlike other apes, human males often engage in extensive investment in their mates and offspring likely further exacerbated this situation via two avenues. First, the risk of misallocation of resources due to cuckoldry further increases the selective advantages of male psychological attributes that deter transgression (Buss, Larsen, Westen, & Semmelroth, 1992; Wilson & Daly, 1992a). Second, with male parental investment a possibility, the ability to obtain and retain resources becomes an important determinant of a man’s attractiveness to women (Buss, 1989). Accordingly, men who can successfully deter transgressions may be more attractive to women by virtue of the physical and economic security they offer.

Together, the above factors are likely to have selected for a sex difference in the subjective response to transgression – because, averaged over time, the stakes to be won or lost in transgressions were considerably higher for men than for women, consistent with dimorphism in size and strength, selection is likely to have favored males who, in comparison with females, were both more easily and more dramatically blinded by anger; the result, tragically, is the male propensity for direct aggression against, and an unwillingness to back down from, the targets of anger. In contrast, in responding to transgression, women frequently adopt lower-risk strategies that rely on political tactics: compared to men, when angry, women are more likely to cry (see Chapter 20 by Fischer and Evers, this volume), a signal that serves to recruit aid from allies (Fessler & Moya, 2009); they are more likely to turn to others for support (reviewed in Kring, 2000) and are more likely to harm the target of their anger through gossip and alliance manipulation (reviewed in Campbell, 2002, pp. 90–94).

21.4 Sex Differences in Non-aggressive Risk Taking

Convergent evidence in support of the argument that an evolutionary history of male intrasexual competition is responsible for the sex difference in violent responses to transgression comes from behavior that is not motivated by anger (Wilson & Daly, 1993; Daly & Wilson, 2001). Risk taking in nonconflictual situations can serve many of the same ends as confrontational risk taking by establishing a reputation for insensitivity to costs (Daly & Wilson, 2001) – competitors will think twice before transgressing against someone who routinely risks life and limb (a benefit that explains the exacerbating effects of an audience on men’s, but not women’s, risk-taking behavior [Daly & Wilson, 2001], as well as the fact that men engage in more recreational risk taking than would be optimal if their sole goal was to impress women [Farthing, 2005]). Hence, because the decision not to walk away from a brewing conflict is a decision to take risks, patterns of nonviolent risk taking

provide additional evidence that the sex difference in homicide participation stems not from a difference in costs but rather from a difference in sensitivity to them (Wilson & Daly, 1993; Daly & Wilson, 2001). For example, controlling for distance driven, at all ages except the elderly, in the United States men are more likely to be involved in fatal automobile accidents than women (Massie, Campbell, & Williams, 1995); tellingly, men are particularly overrepresented in accidents caused by a loss of control, as occurs during risky maneuvers (Tavris, Kuhn, & Layde, 2001). Men are likewise more likely to be involved in auto accidents in Spain (Claret et al., 2003), Thailand (Böhning & Na Ayutha, 1997), Chile (Bedregal et al., 1997), and the Netherlands (Kingma, 1994). U.S. male undergraduates are more likely than their female compatriots to drive, swim, or boat while under the influence of alcohol, and less likely to wear a seat belt (West, Moskal, Dziuban, & Rumbough, 1996). Among all but the elderly, Finnish men are more likely than Finnish women to be injured or killed in an accidental fall (Malmivaara, Heliövaara, Knekt, Reunanen, & Aromaa, 1993), and Dutch men are more likely than Dutch women to be injured in accidental falls, injured by a sharp instrument, and injured by falling objects (Kingma, 1994); likewise, Korean men are more likely than Korean women to suffer injuries requiring hospitalization (Paek, Chun, & Cho, 2007). Lest these patterns be dismissed as artifacts of cultural norms regarding the meaning of driving styles, a gendered division of labor, etc., consider a domain in which no such models apply, the epidemiology of rattlesnake bites: in California, 93% of bite victims are male; this is clearly a product of a sex difference in willingness to approach dangerous animals, as 85% of bites occur on the finger or hand (Wingert & Chan, 1988); similarly, in Arizona, 64% of male bite victims recognized an encounter with a snake but did not attempt to move away, while the same was true of only 9.1% of female victims (Curry et al., 1989). Turning to interactions with another dangerous reptile, 85.4% of the victims of alligator attacks in the United States are male, with “attempting to capture/pick up/exhibit” constituting the commonest precipitating activity (Langley, 2005). Men literally place their hands, and their futures, in death’s jaws.

21.4.1 Aggressiveness and Impulsivity, Constituents of the Male Flash of Anger

In light of its more frequent and more extreme manifestation in men, I term the experience of intense rage in response to transgression that leads to sudden physical aggression *the male flash of anger*. Although it is subjectively experienced as a single, unified event, the male flash of anger probably consists of at least two discrete motivational components. First is *the competitively aggressive component*, the desire to dominate others (including, but not limited to, the desire to promote one’s own interests at others’ expense). In the event of transgression, this orientation manifests as a desire to harm the transgressor. Second is *the risk indifference component*, the willingness to subject oneself to danger in order to promote one’s own interests. In the event of transgression, this orientation manifests as an insensitivity to the potential costs of efforts to harm the transgressor. As noted earlier, indifference to current costs is a core element in anger’s utility as a mechanism to counteract time discounting in protecting against transgression. Additionally, inherent features of the dynamics between actor and transgressor further favor indifference to risk. First, since transgressions are often brief, if a reaction is to effectively truncate a transgression, it must take place quickly. Second, rapid reactions are more effective than delayed reactions at deterring additional transgressions. This is because (a) rapid reactions eliminate a time lag during which further transgressions can occur, and (b) transgressors discount the future relative to the present, thus they will be more dissuaded by the prospect of an immediate reaction than by a delayed reaction of equivalent intensity. Hence, for several reasons, immediate aggressive responses to transgressions are more effective than delayed responses. However, because immediate responses do not allow the actor to control the circumstances

surrounding the action to the same degree as delayed responses, immediate responses generally entail greater risk than delayed responses. The value of immediacy thus further favors risk indifference in reactions to transgressions.

Consistent with the above logic, experimentally induced anger leads men to forgo a guaranteed monetary payout in favor of a risky bet; despite producing the same self-reported intensity of anger in women, this procedure has no effect on women's inclination to gamble (Fessler, Pillsworth, & Flamson, 2004). The male propensity for direct aggression in response to transgression thus reflects the greater male propensity to become blind to costs, and see only benefits, when angry.

21.4.2 Probable Physiological Substrates of the Male Flash of Anger

Although complex psychological phenomena are unlikely to reduce to singular physiological causes, it is useful to consider some likely substrates of the male flash of anger. First, testosterone is clearly associated with dominance behavior (Mazur & Booth, 1998). Moreover, although the picture is complex and the direction of causality not always clear, studies involving normal adults, adolescents, convicted criminals, and animal models suggest a correlation between testosterone and aggressiveness, specifically as regards responses to challenges (i.e., transgression) (Archer, 2006; van Bokhoven et al., 2006; Klinesmith, Kasser, & McAndrew, 2006). Salivary testosterone predicts whether men will pursue additional competition following an initial contest (Mehta & Josephs, 2006); of particular interest, the same measure correlates positively with both the presence of anger and the selective attention to angry faces (van Honk et al., 1999). Next, evidence supports a connection between reduced serotonergic activity and impulsivity in general, and impulsive aggression and/or anger in particular; a number of studies suggest that this connection is more robust in men than in women (see Chapter 6 by Bond and Wingrove, this volume, and Chapter 3 by Reuter, this volume, for reviews).

Experiments in rodents point toward a twofold interaction between androgens and serotonergic activity: First, males exhibit lower brain serotonin levels, particularly in the limbic system (Carlsson & Carlsson, 1988; but see also Haleem, 1992), and experimental androgenization reduces serotonin levels in the amygdala (Sundblad & Eriksson, 1997; but see also Fluegge, Kramer, Rensing, & Fuchs, 1998). Second, androgenization increases lability in serotonergic activity (Cologer-Clifford, Simon, Richter, Smoluk, & Lu, 1999), presumably resulting in substantial behavioral plasticity in males, a possibility addressed below.

21.5 Adaptive Modulation of Risk Taking

In general, individuals who have a rosy future ahead of them should be averse to significant risks, while those who have poorer prospects should be more willing to gamble (Wilson, Daly, & Pound, 2002). Because personal experience constitutes the best basis for predicting one's future, we can expect individuals to be equipped to use past experiences to assess future prospects and to adjust risk-taking behavior in light of this (ongoing) assessment (Hill, Thomson Ross, & Low, 1997). Congruent with this position, highly traumatic experiences can produce post-traumatic stress disorder, in which anger plays a prominent role; reduced serotonergic functioning is implicated in this condition (reviewed in Chapter 6 by Bond and Wingrove, this volume). Experimental modification of rearing conditions in a nonhuman primate model indicates that adverse early experiences result in sub-normal levels of brain serotonin (Rosenblum, Coplan, Friedman, & Bassoff, 1994; Higley & Linnoila, 1997). In humans, exposure to harsh parenting is negatively correlated with the density

of serotonin receptors (Pine et al., 1996), and positively correlated with the risk of later committing murder (Lewis, 1985), being murdered (Allgulander & Nilsson, 2000), and being involved in an automobile accident (Harano, Peck, & McBride, 1975). Among incarcerated adult male violent offenders, recidivism is predicted by low levels of a serotonin metabolite, and this in turn is correlated with a childhood history of paternal alcoholism and violence, paternal absence, and the presence of brothers (who can be construed as competitors) in the home (Virkkunen, Eggert, Rawlings, & Linnoila, 1996). Hence, experiences indicative of a challenging local environment in which the prospects for success are poor appear to cause a decrease in serotonergic activity, predisposing the individual to violence and other forms of risk taking in a manner that, in ancestral populations, would have been adaptive.

21.5.1 Changes Across the Male Life Cycle

In addition to between-individual differences in risk taking that are driven by differences in future prospects, risk taking can be expected to vary across the life cycle as a result of differences in future prospects at different life stages. Moreover, because such within-individual variation in risk taking is likely to be patterned by features of the larger social structure, the timing of relative changes in risk taking will be similar between individuals even though the magnitude of risk taking varies. For most of human history, male social position has probably depended principally upon individual achievement. Considerable time is required to both acquire and demonstrate expertise in socially valued skills, and this was likely at least as true in the past as it is today, if not more so. For example, comparisons among extant foraging societies reveal that expertise in hunting, the principal male economic activity and an important determinant of male social influence, is only achieved after approximately 20 years of learning and practice, i.e., middle-aged men are the experts (Ohtsuka, 1989; Kaplan, Hill, Lancaster, & Hurtado, 2000). Keeping in mind that, consistent with the importance of resources in female reproduction, women highly value social status and access to resources in a prospective mate, consider, therefore, the plight of the adolescent or young adult male: showing outward signs of maturity, and motivated to gain access to the perquisites (including sexual opportunities) of successful older men, young men nevertheless enter the social arena at a competitive disadvantage. Young adulthood thus intrinsically entails a drop in status – even the most popular of boys finds himself near the bottom of the pecking order once he begins to be compared to adult men. At the same time, having few skills and a minimal track record, the young man's future is notably uncertain. Moreover, he is surrounded by age-mates who, facing the same dilemma, constitute both present and future rivals in the competition for status and mates.

Young men have little to lose and much to gain by taking risks, suggesting that men's sensitivity to potential costs will be lowest during young adulthood (Wilson et al., 2002), a prediction supported by the demography of both violent altercations and accidental injuries (Wilson & Daly, 1993; Gardner, 1993). In a variety of contemporary nation-states, young men are both more likely to kill and to be killed than older men (Daly & Wilson, 1990); young men were likewise disproportionately the victims of homicide in historic and prehistoric communities of the indigenous Chumash of California (Lambert, 1994). In the United States, excluding the elderly, young men are more likely to be involved in fatal automobile accidents (Massie et al., 1995) and auto accidents caused by a loss of control (Tavris et al., 2001); similar patterns are evident among Spanish (Claret et al., 2003) and Thai drivers (Böhning & Na Ayutha, 1997); likewise, the median age of rattlesnake bite victims is 22, and 55% of all bites are suffered by patients aged 17–27 (Wingert & Chan, 1988). Consonant with these patterns, the frequency of experiencing, the intensity of, and the likelihood of acting on anger declines with age (reviewed in Chapter 19 by Schieman, this volume).

It is possible to guess at the proximate mechanisms underlying age-related changes in male risk taking. Animal models suggest that social rank is inversely correlated with serotonergic activity, apparently because subordination decreases serotonergic activity, while dominance sometimes enhances it (Dhingra, Lakshmana, Meti, & Raju, 1996; Berton, Durand, Aguerre, Mormède, & Chaouloff, 1999; Westergaard, Suomi, Higley, & Mehlman, 1999). Because testosterone directly affects serotonergic functioning, young males suffer the serotonergic double whammy of simultaneous decreases in status and increases in testosterone, resulting in increases in impulsivity and impulsive aggression. Hence, the typical declines in risk taking and impulsivity that occur over the course of later adulthood may in part be tied to the manner in which age and experience often allow the individual to rise through the social ranks, resulting in (from an ultimate perspective) a decline in the utility of risk taking and (from a proximate perspective) an increase in serotonergic functioning.

In keeping with the match between psychology and morphology noted earlier, patterned changes in sensitivity to risk and propensity to experience anger over the life cycle may be matched by changes in the male body. Observers have noted the correlation in males between the dramatic increases in strength that occur during and following puberty and increased involvement in risk taking, importantly including violence (Wilson & Daly, 1993; Daly & Wilson, 1990). This is understandable in terms of the distribution and composition of male musculature, and the changes therein that occur later in life. First, in young adulthood, males have a large ratio of upper-to-lower-body muscle mass; as men age, this ratio decreases (Gallagher & Heymsfield, 1998). Second, changes occur in the composition of male muscles. Skeletal muscles are composed of two classes of muscle fibers, Type I, or slow twitch fibers, and Type II, or fast twitch fibers. Type I fibers contribute principally to endurance, and Type II fibers, which are metabolically more expensive, contribute principally to power (Fitts, McDonald, & Schluter, 1991; Herbison, Jaweed, & Ditunno, 1982). Beginning sometime in the 20s, there is a decline in both the size of muscles and the number of muscle fibers. This decline disproportionately affects Type II fibers (Kirkendall & Garrett, 1998), particularly in men (Lindle et al., 1997; Neder, Nery, Silva, Andreoni, & Whipp, 1999). Correspondingly, the basal metabolic rate decreases with age, in part due to a reduction in both the quantity of lean tissue and the energetic demands per unit mass of that tissue. Hence, in early adulthood, male muscle distribution is such as to maximize upper body strength, and muscle composition is such as to maximize power. Even at rest, this arrangement is energetically expensive to maintain. Moreover, the combination of a large upper body and a high percentage of easily fatigued muscle fibers greatly limits endurance.

Testosterone increases both muscle size (Bhasin et al., 1996) and the percentage of Type II fibers (Mero, Jaakkola, & Komi, 1991; Krotkiewski, Kral, & Karlsson, 1980). Paralleling age-related changes in muscle distribution and muscle composition, men experience a progressive decline in testosterone levels with age (Harman, Metter, Tobin, Pearson, & Blackman, 2001; Feldman et al., 2002), although there are substantial cross-population differences in the rate of decline, possibly due in part to dietary factors (Ellison & Panter-Brick, 1996; Ellison et al., 1998, 2002; Campbell, Leslie, & Campbell, 2006). In addition to changes in testosterone, decreases in muscle mass with age are associated with declining levels of growth hormone (Harper, 1998; Zaccaria, Varnier, Piazza, Noventa, & Ermolao, 1999), the production of which is stimulated by androgens (Angele, Ayala, Cioffi, Bland, & Chaudry, 1998).

The above findings suggest that adult male development follows a pattern in which, initially, all of the proverbial eggs are put into one basket. Early adulthood is characterized by costly muscles that are most useful in combat, muscles that maximize power at the expense of endurance. At the same time, young men are less sensitive to risk and more predisposed to attend to signs of anger in others, and to experience anger themselves, than older men. This combination leads to violent gambles in an attempt to establish a reputation as someone against whom transgression is costly. The potential costs of this gambit are further elevated by the fact that testosterone, the

proximate cause of enhanced muscular power and aggressiveness, also causes immunosuppression, thus increasing the likelihood that injury will lead to sepsis (Muehlenbein & Bribiescas, 2005; but see also Granger, Booth, & Johnson, 2000); likewise, because serotonin is involved in a variety of immune responses (Mössner & Lesch, 1998), reductions in social status associated with young adulthood are likely to compound the immunosuppressive effects of testosterone. While it might seem maladaptive to diminish immune responses precisely during that phase when trauma is most likely, the immunosuppressive effects of testosterone may be part of the “eggs in one basket” pattern: given that physiological resources are finite, and given that immune responses are energetically expensive, investing in immune responses can be viewed as trading current assets for future health (Muehlenbein & Bribiescas, 2005). However, the more equivocal the future, the less worthwhile such a trade becomes. Accordingly, young males with uncertain futures may generally be better off restricting immune responses in order to maximize the energy available for immediate needs.

The combined costs of increased caloric requirements and heightened vulnerability to pathogens make it difficult to maintain the young male configuration of high upper-to-lower body muscle mass ratio, high Type II-to-Type I muscle fiber ratio, and high-testosterone/low immune response. For successful young men, however, this limitation does not pose an obstacle: after a period of aggressive risk taking, they gradually shift to an alternate strategy, building a reputation based on socially valued skills. In ancestral environments, these skills would have demanded endurance rather than power. Hunting tactics vary markedly between groups, and the energetic demands of hunting are importantly contingent on the techniques employed (Kelly, 1995). Nevertheless, several generalizations apply across hunting techniques. First, because prey animals are likely to avoid encampments, hunting frequently entails extended foot travel; the successful hunter must then transport meat over often-considerable distances. Second, arrows and spears do not readily bring down big game, and hunters typically rely on poison or bleeding to weaken large prey (Kelly, 1995), a time-consuming process that often involves pursuit. Hunting thus likely frequently entails prolonged moderate exercise (see Worthman & Konner, 1987), and hence endurance will often be more of a determinant of success than power. For example, among the hunter-horticulturalist Aché of Paraguay, though stronger than intermediate-sized men, large men likely have poorer endurance, potentially explaining their reduced hunting return rates relative to the latter (Hill & Hurtado, 1996, p. 372).

Changes in male musculature over the life cycle can be seen as both paralleling and facilitating a change in social strategy. Men initially seek to establish a reputation that precludes transgressions; then, as they acquire skills, they scale back their physical confrontations. It is only when their prior reputations are called into question that older men may again resort to combat. However, having acquired social prominence, successful mature men are unlikely to face challengers alone, thus making up for decreases in their fighting abilities (see, for example Hart & Pilling, 1960). Hence, the trajectory of hormonally mediated mental and physical changes is understandable in terms of the varying utility of risk taking and combat during different life phases – risk-prone high-power young men are built for danger and fighting; mellower, high-endurance older men are built for hunting and politics. Lastly, because age-dependent muscular changes are likely to have a panmammalian component, these physiological changes are best viewed as having set the stage for, and perhaps having further coevolved with, the human pattern of changes in strategy over the male life course.²

²Bringing together the threads of the argument made here, Fairbanks et al. (2004) demonstrated that, in adolescent male vervet monkeys, large body size, low serotonergic functioning, and high impulsivity (including impulsive aggression) combine to predict subsequent attainment of alpha status in adulthood. Consonant with a strategic shift in tactics over the life course, though highly impulsive as adolescents, males who reached alpha status decline in impulsivity once they have achieved high rank.

21.5.2 *Reproductive Status and Male Risk Taking*

While important, age is not the only factor expected to influence male competitive strategies. If differences in reproductive success were the engine driving the evolution of the male flash of anger in ancestral populations, and if the psychology of this reaction incorporates an evaluation of present and future prospects, then the extent to which a man of a given age is presently achieving success in the reproductive arena should influence his propensity to respond aggressively to transgression (Wilson & Daly, 1993). Put differently, under ancestral circumstances, whereas a young bachelor whose reproductive future was uncertain was best served by a risk-prone strategy, a married father would have done better to scale back his risk taking lest he lose those gains achieved to date (including losses incurred if, due to his death or injury, his existing dependent offspring were to lose the resources and protection he provides). This suggests that the male flash of anger should decline as a function of marital status and fatherhood.

Consistent with the above logic, there is a striking negative correlation between marital status and participation in male violence. In Canada (Daly & Wilson, 1990) and Florida (West et al., 1996) married men are less likely than same-age single men to commit violence, while in Sweden (Allgulander & Nilsson, 2000) married men are less likely to be killed than single men. Similarly, consistent with the assertion that involvement in violence reflects a willingness to take risks, the same patterns hold true with regard to automobile accidents in the United States (Harano et al., 1975), Thailand (Böhning & Na Ayutha, 1997), and Brazil (Barreto, Swerdlow, Smith, & Higgins, 1997); with regard to both driving under the influence of alcohol and driving without a seat belt in the United States (West et al., 1996), with regard to driving under the influence of cannabis in Canada (Walsh & Mann, 1999), with regard to suffering injurious or fatal falls in Finland (Malmivaara et al., 1993), with regard to all accidental causes of death among Finns (Notkola, Martikainen, & Leino, 1993) and U.S. soldiers (Garvey Wilson, Lange, Brundage, & Frommelt, 2003), and with regard to repeated admission to a hospital emergency room in Ireland (Murphy et al., 1999). The above patterns are consonant with measures of a key proximate factor thought to play a role in competitive male risk taking: with only a few exceptions, studies reveal that married men, and those in committed long-term relationships, have lower salivary testosterone levels than do single men (reviewed in Gray & Campbell, 2009).

While intriguing, correlations such as the above do not allow us to determine the direction of causality – are single men more dangerous because they are single or are they single because they are more dangerous? More specifically, consistent with the future-prospects-based-on-past-experience argument, men with a history of deprivation may well pursue a high-risk strategy aimed at maximizing status and short-term matings; because this is done at the expense of parental investment, such men are less likely to form lasting marriages, or at least less likely to do so early in adult life, than are men from more stable backgrounds who, pursuing lower risk strategies, evince signs of willingness to invest parentally (Belsky, Steinberg, & Draper, 1991; Hill et al., 1997). Two avenues of investigation provide the means to tease apart these possibilities, namely cross-sectional examinations of men who were once married and a unique longitudinal study.

Divorced men are characterized by an increased likelihood of committing murder in the Canadian sample, of being murdered in the Swedish sample, of suffering a fall or other accident in the Finnish samples, of suffering accidental injury requiring hospitalization in a Korean sample (Paek et al., 2007), and of being involved in an automobile accident in three American samples (Harano et al., 1975; McMurray, 1970; Selzer & Vinokur, 1975). These results suggest that risk taking increases when marriages fail. However, because it is possible that these men's marriages failed because they were risk takers, more compelling evidence of such an effect comes from a similar association with widowhood (Wilson & Daly, 1993): compared to married men, widowers are at increased risk of

committing murder in the Canadian sample, of being murdered in the Swedish sample, of being hurt or killed in a fall or other accident in the Finnish and Korean samples, and of being involved in an automobile accident in the U.S. samples. These results suggest that, while some of the contrasts between married and single men may be due to the pursuit of alternate strategies, marriage likely has an independent ameliorating effect on male risk taking.

In the only investigation of its kind, Farrington and West (1995) conducted a longitudinal study of 403 working-class males in London, tracking them from age 8 to 32 to explore the determinants of criminal offending (a class of behaviors that is larger than violent conflict per se, but includes it, as well as other forms of risk taking). Single men were nearly twice as likely to commit a criminal offense as married men. Importantly, this cannot be attributed to preexisting differences in inclinations between the two classes of men as, in the 5 years prior to marriage, men who ultimately married offended at almost exactly the same rate as men who were to remain single. More complexly, an examination of men who married but later separated provides support for both the notion that there are typological differences between men who stay married and men who do not, and the idea that marriage reduces risk taking. During the period in which they were married, men who later separated committed 66% more offenses than men who subsequently remained married. However, like the latter, the former displayed a reduction in offending during the period of marriage relative to the 5 years prior to marriage; most notably, they also exhibited a 44% increase in offending following the end of their marriage, strongly suggesting that marriage reduces risk taking.

From an evolutionary perspective, marriage is not an end in itself, but rather an avenue to reproduction. Accordingly, we should expect that the birth of a child in whom a man intends to invest will be accompanied by a reduction in his participation in violence and other forms of competitive risk taking (note that the qualifying clause is important given that, while investment boosts the likely success of a given child, a viable alternative strategy is to father many children and invest little in each, a trajectory consonant with increased competition with other males, and hence increased risk taking). However, in contrast to work on marital status, surprisingly little research has been done on the effects of fatherhood.³ A number of studies have found that fatherhood is associated with lower testosterone levels (reviewed in Gray & Campbell, 2009); however, as in the case of the hormonal correlates of marriage, in the absence of longitudinal studies, it is not yet possible to determine the direction of causality in these correlations.

To summarize the above, consonant with theoretical predictions, there is reasonable evidence that marriage reduces behaviors of the type associated with the male flash of anger, and there is preliminary evidence that fatherhood may have a similar effect. Recall, however, that these predictions derive from the premise that a pattern of mild polygyny characterized the social worlds in which the human mind evolved. From a reproductive standpoint, in a polygynous environment, it is not optimal to permanently reduce risky competitive behavior following marriage and fatherhood. Rather, male reproductive success is maximized by adjusting such behavior as a function of its costs and benefits. As a woman ages, the number of future offspring that she might bear diminishes to zero; as children mature, the extent to which they benefit from a given unit of paternal investment likewise declines. Correspondingly, for a husband and father, the reproductive benefits of a risk-avoidant strategy decrease over time, hence the pacifying effects of these roles can be expected to exhibit a similar chronology. Specifically, within the constraints of age-related changes in male fighting ability and concomitant risk-taking propensities, the male flash of anger is predicted to exhibit

³Although Farrington and West (1995) evaluated the relationship between fatherhood and criminal offending, they were unable to differentiate the effect of fatherhood within marriage from the effect of marriage itself. Consistent with a strategy of short-term mating, low parental investment, and high risk taking, unmarried fatherhood increased the risk of offending.

periodicity wherein aggressive response to transgression and other forms of risk taking increase as a man's wife's residual reproductive potential diminishes and as his children become more independent. More broadly, the much-debated "male midlife crisis" may reflect such a pattern in culturally monogamous societies (in which polygynous inclinations are maximally disruptive and in which norms prescribe only slight age differences between spouses), with conflicting findings reflecting the fact that it is not principally a man's age that matters, but rather those of his wife and children. Note that increased risk taking behavior in midlife can thus be expected to be a uniquely male phenomenon. While women may experience distress over the fading of their youth, because a woman's attractiveness as a prospective mate is largely age-dependent, women gain little by returning to the behaviors of their single days – whereas men can partially revive their earlier competitive status in the mating arena by altering their behavior, such changes will not suffice for women. Likewise, women with children can be expected to be more risk-averse than women without (due in part to children's vulnerability to retribution, and in part to their dependency on maternal support), but, unlike men, they should not display a return to risk-seeking behaviors as their children mature, since they cannot recapture their earlier mate value, and exposure to risk simply impairs their ability to enhance their fitness through grandmaternal investment. In sum, men, but not women, can be expected to display a rebound in risk taking, including participation in violence motivated by anger, as a function of the ages of their spouse and children.

21.6 Interindividual Variation in Innate Propensities

The experience of the male flash of anger and its life course vicissitudes are predicted to be universal. However, universality is not the same as uniformity, and there is substantial evidence of heritable variation in the psychological traits, and their likely physiological substrates, that compose the male flash of anger. Considerable evidence supports the heritability of a propensity toward anger and aggression (reviewed in Chapter 3 by Reuter, this volume). Consistent with these findings, heredity is a significant determinant of testosterone levels (Kuijper et al., 2007), and polymorphisms largely responsible for interindividual variation in the degree of age-related decline in testosterone levels have been identified (Krithivas et al., 1999). Likewise, serotonergic functioning has a substantial heritable component (see Chapter 3 by Reuter, this volume; Jabbi et al., 2007).

The genetic determinants of the male flash of anger are most likely complex, involving multiple component processes. This apparently results in numerous avenues that can lead to inter-individual variation in the propensity to react violently to transgression. Given the sometimes substantial fitness consequences of reacting in accord with, or refraining from, the male flash of anger, it is not implausible that the heritable variation described above has, or had, long-term functional significance.

21.6.1 *Culture and Genetic Variation in the Male Flash of Anger*

Although inter-individual differences in the propensity to respond to transgression with violent anger are readily observable, some of the most dramatic differences in attitudes toward, and incidences of, male violence occurs not between individuals but between groups (Ghiglieri, 1999). Cultural anthropologists have long argued that the difference between "warlike" and "peaceful" societies derives from the meaning attached to violence itself (Robarchek & Robarchek, 1992), an argument that can be extended to include the cultural construal of anger (Myers, 1988; Johnson et al., 1986; Levy, 1973;

Fessler, 2006) and the very meaning of transgression (Nisbett & Cohen, 1996). These cultural factors may interdigitate with the determinants of aggressiveness discussed thus far in several ways. First, by shaping the environment of childhood experience, cultural factors may influence perceptions of future prospects that influence levels of risk sensitivity (Belsky et al., 1991). Second, by favoring greater or lesser degrees of aggressive risk-indifferent response to transgression, culture could conceivably constitute a source of selection operating on heritable variation in factors contributing to this trait. Of these two possibilities, the latter is far more controversial, as it suggests that individuals descended from groups having differing orientations toward violent response to transgression will differ in their baseline propensities for such behavior.

I know of no reliable data that bear directly on the question of between-population differences in the heritable components of the male flash of anger. However, other findings suggest that such differences could occur. First, culturally shaped dietary practices can modify heritable attributes: pastoralists exhibit high levels of adult lactase production, tropical populations without ready access to salt may be characterized by high sodium retention, and arctic hunters with low plant food intake efficiently produce glucose from amino acids (Lieberman, 1987). Second, findings concerning the frequency of polymorphisms of a dopamine receptor gene suggest that cultural practices may select for heritable personality traits, as a variant associated with sensation seeking is more common in pastoralists (who must be mobile) than in agriculturalists (who are sedentary) (Chen, Burton, Greenberger, & Dmitrieva, 1999). It is thus conceivable that cultural traditions that pre- or proscribe violent response to transgression might have similar effects with regard to heritable aspects of the male flash of anger. However, a number of factors raise doubts as to the likelihood of this.

In order for cultural beliefs and practices to shape frequencies of alleles contributing to the male flash of anger, those aspects of a given culture must remain constant for considerable periods of time. Although documented cases of such longevity exist (cf. Nisbett & Cohen, 1996), it is also known that cultures can change rapidly, particularly in cases of culture contact and/or assimilation (Levy, 1973; Tuzin, 1989, pp. 187–208). Contrary to stereotypes of pristine, isolated cultural traditions in small-scale societies, it is likely that diffusion and acculturation were the norm, rather than the exception, in human history. Moreover, it is not merely ideas that are exchanged between groups, but genes as well, and hence any homogenizing effects of a given culture are likely to be diluted through contact with other groups. Lastly, even in small-scale societies having a relatively monolithic approach toward anger and violence, there are often multiple pathways to social success, with repeated confrontational aggression being only one avenue (cf. Chagnon, 1997); the same may be less true of subsistence practices, and hence the cases discussed above may not generalize to other domains.

Cross-population studies of the frequencies of genetic contributors to the male flash of anger can potentially shed light on the question of the influence of culture on heritable dispositions. However, the liabilities entailed by such research are considerable given the popular media's incomplete reporting of scientific findings – often, although the same study that demonstrates heritability also illuminates how environmental conditions profoundly shape development, only the former is highlighted. Investigators must therefore exercise caution in approaching this question lest they contribute to the formation or perpetuation of racist stereotypes.

21.7 Moral Outrage: A Uniquely Human Form of Anger

Although it is questionable whether specific cultural traditions have constituted sources of selective pressure shaping the heritable substrates of anger phenomena, the relationship between culture writ large and human responses to transgression is more clear-cut. While a variety of mammals and birds

possess rudimentary cultural norms governing behavior, our species is unique not only in the richness and variety of such norms but, more importantly, in the fact that individual actors enforce norms the violations of which do not affect them. From an evolutionary perspective, the latter is puzzling – all else being equal, we might expect that fitness would be reduced by incurring costs (time, energy, social capital, and risk of injury or retribution) to enforce rules that do not bear directly on one's own welfare. Several possible explanations have been advanced (reviewed in Hagen & Hammerstein, 2006). The propensity to engage in costly norm enforcement may have been favored by any of several forms of evolutionary group selection, as groups that, via such enforcement, functioned effectively would have out-competed less well-organized rivals, leading to the prevalence of the propensity to enforce. Alternately, norm enforcement may serve a communicative function, as policing others' behavior offers an opportunity to demonstrate that the enforcer shares prevailing cultural values and acts in accord with them, even at a cost to the self, attributes that make the actor attractive as an ally or member of a cooperative venture; in turn, these roles may entail benefits that outweigh the costs of policing.

A principal emotion motivating punishment of norm violators is a form of anger – phenomenologically and behaviorally, many people respond to transgressions against norms as if they constituted transgressions against the self. Because the eliciting conditions are different from that of simple anger and because the evolutionary function of the emotion necessarily differs from that of simple anger, this emotion can be usefully distinguished using the term *moral outrage* (Fessler & Haley, 2003). Evolutionary processes frequently involve modifying existing mechanisms in order to address new challenges, and moral outrage can be seen as such a modification. Importantly, whereas we can expect all complex social animals to experience something like anger, given the unique role of culture in human social behavior, the same is not true of moral outrage. Investigations of the determinants of moral outrage offer an avenue for testing the signaling hypothesis outlined above, as, being premised on reputational issues, this perspective generates predictions paralleling those for simple anger (Fessler & Haley, 2003). Briefly, we should expect the presence of an audience to increase moral outrage. Likewise, willingness to take risks in order to inflict costs on norm violators should be a function of both the level of competition and the potential benefits of establishing a positive reputation – morally outraged men can be expected to be more risk-prone than their female counterparts, and young men, who are entering the political arena for the first time, should be the most willing to take such risks. As the case of moral outrage illustrates, applying functionalist evolutionary reasoning is a productive source of hypotheses concerning the male flash of anger.

Acknowledgments This essay owes much to the pioneering and authoritative work of Martin Daly and Margo Wilson. I am grateful to Mike Potegal for useful comments and the opportunity to contribute to this volume, and to Rob Boyd, Joe Manson, Nick Blurton-Jones, Eric A. Smith, and Margo Wilson for productive discussions.

References

- Allgulander, C., & Nilsson, B. (2000). Victims of criminal homicide in Sweden: A matched case-control study of health and social risk factors among all 1,739 cases during 1978–1994. *American Journal of Psychiatry*, 157(2), 244–247.
- Angele, M. K., Ayala, A., Cioffi, W. G., Bland, K. I., & Chaudry, I. H. (1998). Testosterone: The culprit for producing splenocyte immune depression after trauma hemorrhage. *American Journal of Physiology*, 274(6 Part 1), C1530–C1536.
- Archer, J. (2006). Testosterone and human aggression: An evaluation of the challenge hypothesis. *Neuroscience and Biobehavioral Reviews*, 30(3), 319–345.
- Bailey, J. E., Kellermann, A. L., Somes, G. W., Banton, J. G., Rivara, F. P., & Rushforth, N. P. (1997). Risk factors for violent death of women in the home. *Archives of Internal Medicine*, 157(7), 777–782.

- Barreto, S. M., Swerdlow, A. J., Smith, P. G., & Higgins, C. D. (1997). Risk of death from motor-vehicle injury in Brazilian steelworkers: A nested case-control study. *International Journal of Epidemiology*, 26(4), 814–821.
- Bedregal, P., Romero, M. I., Bastías, G., Castillo, J., Pinto, J. I., & Benavides, E. (1997). Mortalidad por accidentes de tránsito en Chile, 1994: una aproximación epidemiológica. *Revista Medica de Chile*, 125(9), 1097–1102.
- Belsky, J., Steinberg, L., & Draper, P. (1991). Childhood experience, interpersonal development, and reproductive strategy: An evolutionary theory of socialization. *Child Development*, 62(4), 647–670.
- Berton, O., Durand, M., Aguerre, S., Mormède, P., & Chaouloff, F. (1999). Behavioral, neuroendocrine and serotonergic consequences of single social defeat and repeated fluoxetine pretreatment in the Lewis rat strain. *Neuroscience*, 92(1), 327–341.
- Bhasin, S., Storer, T. W., Berman, N., Callegari, C., Clevenger, B., Phillips, J., et al. (1996). The effects of supra-physiologic doses of testosterone on muscle size and strength in normal men. *New England Journal of Medicine*, 335(1), 1–7.
- Böhning, D., & Na Ayutha, R. S. (1997). A case-control study of non-fatal traffic accidents on hospital patients in Bangkok metropolis. *Sozial- und Präventivmedizin*, 42(6), 351–357.
- Bond, A. J., & Wingrove, J. (2010). The neurochemistry and psychopharmacology of anger. In M. Potegal, G. Stemmler, & C. D. Spielberger (Eds.), *Handbook of anger* (pp. 79–102). New York: Springer.
- Burbank, V. K. (1994). *Fighting women: Anger and aggression in Aboriginal Australia*. Berkeley, CA: University of California Press.
- Buss, D. M. (1989). Sex differences in human mate preferences: Evolutionary hypotheses tested in 37 cultures. *Behavioral and Brain Sciences*, 12(1), 1–49.
- Buss, D. M., Larsen, R. J., Westen, D., & Semmelroth, J. (1992). Sex differences in jealousy: Evolution, physiology, and psychology. *Psychological Science*, 3(4), 251–255.
- Campbell, A. (2002). *A mind of her own: The evolutionary psychology of women*. New York: Oxford University Press.
- Campbell, B., Leslie, P., & Campbell, K. (2006). Age-related changes in testosterone and SHBG among Turkana males. *American Journal of Human Biology*, 18(1), 71–82.
- Carlsson, M., & Carlsson, A. (1988). A regional study of sex differences in rat brain serotonin. *Progress in Neuro-Psychopharmacology & Biological Psychiatry*, 12(1), 53–61.
- Chagnon, N. A. (1997). *Yanomamö* (5th ed.). Fort Worth, TX: Harcourt Brace College Publishers.
- Chen, C., Burton, M., Greenberger, E., & Dmitrieva, J. (1999). Population migration and the variation of dopamine D4 receptor (DRD4) allele frequencies around the globe. *Evolution and Human Behavior*, 20(5), 309–324.
- Claret, P. L., Castillo Jde, D., Moleon, J. J., Cavanillas, A. B., Martin, M. G., & Vargas, R. G. (2003). Age and sex differences in the risk of causing vehicle collisions in Spain, 1990 to 1999. *Accident Analysis and Prevention*, 35(2), 261–272.
- Cologer-Clifford, A., Simon, N. G., Richter, M. L., Smoluk, S. A., & Lu, S. (1999). Androgens and estrogens modulate 5-HT1A and 5-HT1B agonist effects on aggression. *Physiology and Behavior*, 65(4–5), 823–828.
- Curry, S. C., Horning, D., Brady, P., Requa, R., Kunkel, D. B., & Vance, M. V. (1989). The legitimacy of rattlesnake bites in central Arizona. *Annals of Emergency Medicine*, 18(6), 658–663.
- Daly, M., & Wilson, M. (1988). *Homicide*. New York: Aldine de Gruyter.
- Daly, M., & Wilson, M. (1990). Killing the competition: Female/female and male/male homicide. *Human Nature*, 1(1), 81–107.
- Daly, M., & Wilson, M. (2001). Risk-taking, intrasexual competition, and homicide. *Nebraska Symposium on Motivation*, 47, 1–36.
- Dhingra, N. K., Lakshmana, M. K., Meti, B. L., & Raju, T. R. (1996). Subordination induced decrease in 5-hydroxytryptamine and dopamine levels in the frontal cortex – A study using worker-parasite relationship in rats as a model. *Indian Journal of Physiology and Pharmacology*, 40(3), 213–219.
- Edwards, D. C. (1999). *Motivation and emotion: Evolutionary, physiological, cognitive, and social influences*. Thousand Oaks, CA: Sage Publications, Inc.
- Ekman, P. (1994). Strong evidence for universals in facial expressions: A reply to Russell's mistaken critique. *Psychological Bulletin*, 115(2), 268–287.
- Ellis, L. (1995). Dominance and reproductive success among nonhuman animals: A cross-species comparison. *Ethology & Sociobiology*, 16(4), 257–333.
- Ellison, P. T., Bribiescas, R. G., Bentley, G. R., Campbell, B. C., Lipson, S. F., Panter-Brick, C., et al. (2002). Population variation in age-related decline in male salivary testosterone. *Human Reproduction*, 17(12), 3251–3253.
- Ellison, P. T., Lipson, S. F., Bribiescas, R. G., Bentley, G. R., Campbell, B. C., & Panter-Brick, C. (1998). Inter- and intra-population variation in the pattern of male testosterone by age. *American Journal of Physical Anthropology*, 105(Suppl. 26), 80.

- Ellison, P. T., & Panter-Brick, C. (1996). Salivary testosterone levels among Tamang and Kami males of central Nepal. *Human Biology*, 68(6), 955–965.
- Fairbanks, L. A., Jorgensen, M. J., Huff, A., Blau, K., Hung, Y. Y., & Mann, J. J. (2004). Adolescent impulsivity predicts adult dominance attainment in male vervet monkeys. *American Journal of Primatology*, 64(1), 1–17.
- Farrington, D. P., & West, D. J. (1995). Effects of marriage, separation, and children on offending by adult males. In Z. S. Blau & J. Hagan (Eds.), *Current perspectives on aging and the life cycle* (Vol. 4, pp. 249–281). Greenwich, CT: JAI Press.
- Farthing, G. (2005). Attitudes toward heroic and nonheroic physical risk takers as mates and as friends. *Evolution and Human Behavior*, 26(2), 171–185.
- Feldman, H. A., Longcope, C., Derby, C. A., Johannes, C. B., Araujo, A. B., Coviello, A. D., et al. (2002). Age trends in the level of serum testosterone and other hormones in middle-aged men: Longitudinal results from the Massachusetts male aging study. *Journal of Clinical Endocrinology and Metabolism*, 87(2), 589–598.
- Felson, R. B. (1996). Big people hit little people: Sex differences in physical power and interpersonal violence. *Criminology*, 34(3), 433–452.
- Fessler, D. M. T. (2001). Emotions and cost/benefit assessment: The role of shame and self-esteem in risk taking. In R. Selten & G. Gigerenzer (Eds.), *Bounded rationality: The adaptive toolbox* (pp. 191–214). Cambridge: MIT University Press.
- Fessler, D. M. T. (2006). Violent response to transgression as an example of the intersection of evolved psychology and culture. In J. Barkow (Ed.), *Missing the revolution: Darwinism for social scientists* (pp. 101–117). New York: Oxford University Press.
- Fessler, D. M. T., & Haley, K. J. (2003). The strategy of affect: Emotions in human cooperation. In P. Hammerstein (Ed.), *The genetic and cultural evolution of cooperation* (pp. 7–36). Cambridge: MIT Press.
- Fessler, D. M. T., & Moya, C. M. (2009). Crying. In K. Scherer & D. Sander (Eds.), *The Oxford companion to the affective sciences* (pp. 105–106). Oxford: Oxford University Press.
- Fessler, D. M. T., Pillsworth, E. G., & Flanson, T. J. (2004). Angry men and disgusted women: An evolutionary approach to the influence of emotions on risk taking. *Organizational Behavior and Human Decision Processes*, 95(1), 107–123.
- Fischer, A. H., & Evers, C. (2010). Anger in the context of gender. In M. Potegal, G. Stemmler, & C. D. Spielberger (Eds.), *Handbook of anger* (pp. 349–360). New York: Springer.
- Fitts, R. H., McDonald, K. S., & Schluter, J. M. (1991). The determinants of skeletal muscle force and power: Their adaptability with changes in activity pattern. *Journal of Biomechanics*, 24(Suppl. 1), 111–122.
- Fluegge, G., Kramer, M., Rensing, S., & Fuchs, E. (1998). 5HT1A-receptors and behaviour under chronic stress: Selective counteraction by testosterone. *European Journal of Neuroscience*, 10(8), 2685–2693.
- Foster, L. A., Veale, C. M., & Fogel, C. I. (1989). Factors present when battered women kill. *Issues in Mental Health Nursing*, 10(3–4), 273–284.
- Fox, J. A., & Zawitz, M. W. (2000). *Homicide trends in the United States*. Washington, DC: Bureau of Justice Statistics (10/6/00).
- Frank, R. H. (1988). *Passions within reason: The strategic role of the emotions*. New York: W. W. Norton.
- Frederick, S., Loewenstein, G., & O'Donoghue, T. (2002). Time discounting and time preference: A critical review. *Journal of Economic Literature*, 40(2), 351–401.
- Gallagher, D., & Heymsfield, S. B. (1998). Muscle distribution: Variations with body weight, gender, and age. *Applied Radiation and Isotopes*, 49(5–6), 733–734.
- Gardner, W. (1993). A life-span rational-choice theory of risk taking. In N. J. Bell, & R. W. Bell (Eds.), *Adolescent risk taking* (pp. 66–83). Newbury Park, CA: Sage Publications, Inc.
- Gartner, R. (1990). The victims of homicide: A temporal and cross-national comparison. *American Sociological Review*, 55(1), 92–106.
- Garvey Wilson, A. L., Lange, J. L., Brundage, J. F., & Frommelt, R. A. (2003). Behavioral, demographic, and prior morbidity risk factors for accidental death among men: A case-control study of soldiers. *Preventive Medicine*, 36(1), 124–130.
- Gaulin, S. J., & Boster, J. S. (1992). Human marriage systems and sexual dimorphism in stature. *American Journal of Physical Anthropology*, 89(4), 467–475.
- Ghiglieri, M. P. (1999). *The dark side of man: Tracing the origins of male violence*. Reading, MA: Perseus Books.
- Gladwin, T., & Sarason, S. B. (1953). *Truk: Man in paradise*. New York: Wenner-Gren Foundation for Anthropological Research.
- Granger, D. A., Booth, A., & Johnson, D. R. (2000). Human aggression and enumerative measures of immunity. *Psychosomatic Medicine*, 62(4), 583–590.

- Gray, P. B., & Campbell, B. C. (2009). Human male testosterone, pair bonding and fatherhood. In P. B. Gray & P. T. Ellison (Eds.), *Endocrinology of social relationships* (pp. 270–293). Cambridge: Harvard University Press.
- Hagen, E. H., & Hammerstein, P. (2006). Game theory and human evolution: A critique of some recent interpretations of experimental games. *Theoretical Population Biology*, 69(3), 339–348.
- Haidt, J., & Keltner, D. (1999). Culture and facial expression: Open-ended methods find more expressions and a gradient of recognition. *Cognition & Emotion*, 13(3), 225–266.
- Haleem, D. J. (1992). Sex differences in neurochemical and behavioural effects of 8-hydroxy-2-(di-n-propylamino) tetralin. *Life Sciences*, 50(24), PL221–PL226.
- Harano, R. M., Peck, R. C., & McBride, R. S. (1975). The prediction of accident liability through biographical data and psychometric tests. *Journal of Safety Research*, 7(1), 16–52.
- Harman, S. M., Metter, E. J., Tobin, J. D., Pearson, J., & Blackman, M. R. (2001). Longitudinal effects of aging on serum total and free testosterone levels in healthy men. Baltimore Longitudinal Study of Aging. *Journal of Clinical Endocrinology and Metabolism*, 86(2), 724–731.
- Harper, E. J. (1998). Changing perspectives on aging and energy requirements: Aging, body weight and body composition in humans, dogs and cats. *Journal of Nutrition*, 128(12 Suppl.), 2627S–2631S.
- Hart, C. W. M., & Pilling, A. R. (1960). *The Tiwi of North Australia*. New York: Holt.
- Henrich, J., & Gil-White, F. J. (2001). The evolution of prestige: Freely conferred deference as a mechanism for enhancing the benefits of cultural transmission. *Evolution and Human Behavior*, 22(3), 165–196.
- Herbison, G. J., Jaweed, M. M., & Ditunno, J. F. (1982). Muscle fiber types. *Archives of Physical Medicine and Rehabilitation*, 63(5), 227–230.
- Higley, J. D., & Linnoila, M. (1997). Low central nervous system serotonergic activity is trait like and correlates with impulsive behavior: A nonhuman primate model investigating genetic and environmental influences on neurotransmission. In D. M. Stoff, & J. J. Mann (Eds.), *The neurobiology of suicide from the bench to the clinic* (pp. 39–56). New York: New York Academy of Sciences.
- Hill, K., & Hurtado, A. M. (1996). *Aché life history: The ecology and demography of a foraging people*. New York: Aldine de Gruyter.
- Hill, E. M., Thomson Ross, L., & Low, B. S. (1997). The role of future unpredictability in human risk-taking. *Human Nature*, 8(4), 287–325.
- Hirshleifer, J. (1987). On emotions as guarantors of threats and promises. In J. Dupre (Ed.), *The latest on the best* (pp. 307–326). Cambridge: MIT Press.
- Jabbi, M., Korf, J., Kema, I. P., Hartman, C., van der Pompe, G., Minderaa, R. B., et al. (2007). Convergent genetic modulation of the endocrine stress response involves polymorphic variations of 5-HTT, COMT and MAOA. *Molecular Psychiatry*, 12(5), 483–490.
- Johnson, A., Johnson, O., & Baksh, M. (1986). The colors of emotions in Machiguenga. *American Anthropologist*, 88(3), 674–681.
- Kaplan, H., Hill, K., Lancaster, J., & Hurtado, A. M. (2000). A theory of human life history evolution: Diet, intelligence, and longevity. *Evolutionary Anthropology*, 9(4), 156–185.
- Kellermann, A. L., & Mercy, J. A. (1992). Men, women, and murder – Gender-specific differences in rates of fatal violence and victimization. *Journal of Trauma*, 33(1), 1–5.
- Kellermann, A. L., Somes, G., Rivara, F. P., Lee, R. K., & Banton, J. G. (1998). Injuries and deaths due to firearms in the home. *Journal of Trauma*, 45(2), 263–267.
- Kelly, R. L. (1995). *The foraging spectrum: Diversity in hunter-gatherer lifeways*. Washington, DC: Smithsonian Institution Press.
- Kingma, J. (1994). The young male peak in different categories of trauma victims. *Perceptual & Motor Skills*, 79(2), 920–922.
- Kirkendall, D. T., & Garrett, W. E. (1998). The effects of aging and training on skeletal muscle. *American Journal of Sports Medicine*, 26(4), 598–602.
- Klinesmith, J., Kasser, T., & McAndrew, F. T. (2006). Guns, testosterone, and aggression: An experimental test of a mediational hypothesis. *Psychological Science*, 17(7), 568–571.
- Kring, A. M. (2000). Gender and anger. In A. H. Fischer (Ed.), *Gender and emotions* (pp. 211–231). New York: Cambridge University Press.
- Krithivas, K., Yurgalevitch, S. M., Mohr, B. A., Wilcox, C. J., Batter, S. J., Brown, M., et al. (1999). Evidence that the CAG repeat in the androgen receptor gene is associated with the age-related decline in serum androgen levels in men. *Journal of Endocrinology*, 162(1), 137–142.
- Krotkiewski, M., Kral, J. G., & Karlsson, J. (1980). Effects of castration and testosterone substitution on body composition and muscle metabolism in rats. *Acta Physiologica Scandinavica Acta Physiol Scand*, 109(3), 233–237.

- Kuijper, E. A., Lambalk, C. B., Boomsma, D. I., van der Sluis, S., Blankenstein, M. A., de Geus, E. J., et al. (2007). Heritability of reproductive hormones in adult male twins. *Human Reproduction*, 22(8), 2153–2159.
- Lambert, P. M. (1994). *War and peace on the western front: A study of violent conflict and its correlates in prehistoric hunter-gatherer societies of coastal southern California*. Santa Barbara, CA: University of California.
- Langley, R. L. (2005). Alligator attacks on humans in the United States. *Wilderness and Environmental Medicine*, 16(3), 119–124.
- Lee, R. B. (1993). *The Dobe Ju/'hoansi* (2nd ed.). Fort Worth, TX: Harcourt Brace College Publishers.
- Levy, R. I. (1973). *Tahitians: Mind and experience in the Society Islands*. Chicago: University of Chicago Press.
- Lewis, D. O. (1985). Biopsychosocial characteristics of children who later murder: A prospective study. *American Journal of Psychiatry*, 142(10), 1161–1167.
- Lieberman, L. S. (1987). Biocultural consequences of animals versus plants as sources of fats, proteins, and other nutrients. In M. Harris & E. B. Ross (Eds.), *Food and evolution: Toward a theory of human food habits* (pp. 225–258). Philadelphia, PA: Temple University Press.
- Lindle, R. S., Metter, E. J., Lynch, N. A., Fleg, J. L., Fozard, J. L., Tobin, J., et al. (1997). Age and gender comparisons of muscle strength in 654 women and men aged 20–93 yr. *Journal of Applied Physiology*, 83(5), 1581–1587.
- Malmivaara, A., Heliövaara, M., Knekt, P., Reunanen, A., & Aromaa, A. (1993). Risk factors for injurious falls leading to hospitalization or death in a cohort of 19,500 adults. *American Journal of Epidemiology*, 138(6), 384–394.
- Massie, D. L., Campbell, K. L., & Williams, A. F. (1995). Traffic accident involvement rates by driver age and gender. *Accident Analysis and Prevention*, 27(1), 73–87.
- Mazur, A., & Booth, A. (1998). Testosterone and dominance in men. *Behavioral and Brain Sciences*, 21(3), 353–397.
- McGuire, M. T., & Troisi, A. (1990). Anger: An evolutionary view. In R. Plutchik & H. Kellerman (Eds.), *Emotion, psychopathology, and psychotherapy* (Vol. 5, pp. 43–57). San Diego, CA: Academic Press, Inc.
- McMurray, L. (1970). Emotional stress and driving performance: The effect of divorce. *Behavioral Research in Highway Safety*, 1(2), 100–114.
- Mehta, P. H., & Josephs, R. A. (2006). Testosterone change after losing predicts the decision to compete again. *Hormones and Behavior*, 50(5), 684–692.
- Mero, A., Jaakkola, L., & Komi, P. V. (1991). Relationships between muscle fibre characteristics and physical performance capacity in trained athletic boys. *Journal of Sports Sciences*, 9(2), 161–171.
- Mössner, R., & Lesch, K. P. (1998). Role of serotonin in the immune system and in neuroimmune interactions. *Brain, Behavior, and Immunity*, 12(4), 249–271.
- Muehlenbein, M. P., & Bribiescas, R. G. (2005). Testosterone-mediated immune functions and male life histories. *American Journal of Human Biology*, 17(5), 527–558.
- Murdock, G. P. (1967). *Ethnographic atlas*. Pittsburgh, PA: University of Pittsburgh Press.
- Murphy, A. W., Leonard, C., Plunkett, P. K., Brazier, H., Conroy, R., Lynam, F., et al. (1999). Characteristics of attenders and their attendances at an urban accident and emergency department over a one year period. *Journal of Accident and Emergency Medicine*, 16(6), 425–427.
- Myers, F. R. (1988). The logic and meaning of anger among Pintupi Aborigines. *Man*, 23(4), 589–610.
- Neder, J. A., Nery, L. E., Silva, A. C., Andreoni, S., & Whipp, B. J. (1999). Maximal aerobic power and leg muscle mass and strength related to age in non-athletic males and females. *European Journal of Applied Physiology and Occupational Physiology*, 79(6), 522–530.
- Nisbett, R. E., & Cohen, D. (1996). *Culture of honor: The psychology of violence in the South*. Boulder, CO: Westview Press.
- Notkola, V. J., Martikainen, P., & Leino, P. I. (1993). Time trends in mortality in forestry and construction workers in Finland 1970–85 and impact of adjustment for socioeconomic variables. *Journal of Epidemiology and Community Health*, 47(3), 186–191.
- Ohtsuka, R. (1989). Hunting activity and aging among the Gidra Papuans: A biobehavioral analysis. *American Journal of Physical Anthropology*, 80(1), 31–40.
- Paek, K. W., Chun, K. H., & Cho, J. P. (2007). Risk factors influencing the occurrence of injuries in Koreans requiring hospitalization. *Yonsei Medical Journal*, 48(2), 164–170.
- Pine, D. S., Wasserman, G. A., Coplan, J., Fried, J. A., Huang, Y.-Y., Kassir, S., et al. (1996). Platelet serotonin 2A (5-HT-sub(2A)) receptor characteristics and parenting factors for boys at risk for delinquency: A preliminary report. *American Journal of Psychiatry*, 153(4), 538–544.
- Plavcan, J. M. (2001). Sexual dimorphism in primate evolution. *American Journal of Physical Anthropology*, 116(S33), 25–53.
- Reuter, M. (2010). Population and molecular genetics of anger and aggression: Current state of the art. In M. Potegal, G. Stemmler, & C. D. Spielberger (Eds.), *Handbook of anger* (pp. 27–38). New York: Springer.

- Robarchek, C. A., & Robarchek, C. J. (1992). Cultures of war and peace: A comparative study of Waorani and Semai. In J. Silverberg & J. P. Gray (Eds.), *Aggression and peacefulness in humans and other primates* (pp. 189–213). New York: Oxford University Press.
- Rosenblum, L. A., Coplan, J. D., Friedman, S., & Bassoff, T. (1994). Adverse early experiences affect noradrenergic and serotonergic functioning in adult primates. *Biological Psychiatry*, 35(4), 221–227.
- Schelling, T. C. (1980). *The strategy of conflict*. Cambridge: Harvard University Press.
- Schieman, S. (2010). The sociological study of anger: Basic social patterns and contexts. In M. Potegal, G. Stemmler, & C. D. Spielberger (Eds.), *Handbook of anger* (pp. 329–348). New York: Springer.
- Sell, A., Tooby, J., & Cosmides, L. (2009). Formidability and the logic of human anger. *Proceedings of the National Academy of Sciences*, 106(35), 15073–15078.
- Selzer, M. L., & Vinokur, A. (1975). Role of life events in accident causation. *Mental Health & Society*, 2(1–2), 36–54.
- Sundblad, C., & Eriksson, E. (1997). Reduced extracellular levels of serotonin in the amygdala of androgenized female rats. *European Neuropsychopharmacology*, 7(4), 253–259.
- Tavris, D. R., Kuhn, E. M., & Layde, P. M. (2001). Age and gender patterns in motor vehicle crash injuries: Importance of type of crash and occupant role. *Accident Analysis and Prevention*, 33(2), 167–172.
- Tooby, J., Cosmides, L., & Price, M. E. (2006). Cognitive adaptations for n-person exchange: The evolutionary roots of organizational behavior. *Managerial and Decision Economics*, 27, 103–129.
- Tuzin, D. (1989). Visions, prophecies, and the rise of Christian Consciousness. In G. H. Herdt & M. Stephen (Eds.), *The religious imagination in New Guinea* (pp. 187–208). New Brunswick, NJ: Rutgers University Press.
- van Bokhoven, I., van Goozen, S. H., van Engeland, H., Schaal, B., Arseneault, L., Seguin, J. R., et al. (2006). Salivary testosterone and aggression, delinquency, and social dominance in a population-based longitudinal study of adolescent males. *Hormones and Behavior*, 50(1), 118–125.
- van Honk, J., Tuiten, A., Verbaten, R., van den Hout, M., Koppeschaar, H., Thijssen, J., et al. (1999). Correlations among salivary testosterone, mood, and selective attention to threat in humans. *Hormones and Behavior*, 36(1), 17–24.
- Van Kleef, G. A. (2010). Don't worry, be angry? Effects of anger on feelings, thoughts, and actions in conflict and negotiation. In M. Potegal, G. Stemmler, & C. D. Spielberger (Eds.), *Handbook of anger* (pp. 545–560). New York: Springer.
- Virkkunen, M., Eggert, M., Rawlings, R., & Linnoila, M. (1996). A prospective follow-up study of alcoholic violent offenders and fire setters. *Archives of General Psychiatry*, 53(6), 523–529.
- Walsh, G. W., & Mann, R. E. (1999). On the high road: Driving under the influence of cannabis in Ontario. *Canadian Journal of Public Health. Revue Canadienne de Sante Publique*, 90(4), 260–263.
- West, G. B., Moskal, P. D., Dziuban, C. D., & Rumbough, L. P. (1996). Gender and marital differences of risk taking among undergraduates. *Psychological Reports*, 78(1), 315–320.
- Westergaard, G. C., Suomi, S. J., Higley, J. D., & Mehlman, P. T. (1999). CSF 5-HIAA and aggression in female macaque monkeys: Species and interindividual differences. *Psychopharmacology (Berlin)*, 146(4), 440–446.
- Wilson, M., & Daly, M. (1992a). The man who mistook his wife for a chattel. In J. H. Barkow, L. Cosmides, & J. Tooby (Eds.), *The adapted mind* (pp. 289–322). New York: Oxford University Press.
- Wilson, M. I., & Daly, M. (1992b). Who kills whom in spouse killings – On the exceptional sex ratio of spousal homicides in the United States. *Criminology*, 30(2), 189–215.
- Wilson, M., & Daly, M. (1993). Lethal confrontational violence among young men. In N. J. Bell & R. W. Bell (Eds.), *Adolescent risk taking* (pp. 84–106). Newbury Park, CA: Sage Publications, Inc.
- Wilson, M., Daly, M., & Pound, N. (2002). An evolutionary psychological perspective on the modulation of competitive confrontation and risk taking. In D. W. Pfaff, A. P. Arnold, A. M. Etgen, S. E. Fahrbach, & R. T. Rubin (Eds.), *Hormones, brain and behavior* (Vol. 5, pp. 381–408). San Diego, CA: Academic Press.
- Wingert, W. A., & Chan, L. (1988). Rattlesnake bites in southern California and rationale for recommended treatment. *Western Journal of Medicine*, 148(1), 37–44.
- Worthman, C. M., & Konner, M. J. (1987). Testosterone levels change with subsistence hunting effort in !Kung San men. *Psychoneuroendocrinology*, 12(6), 449–458.
- Zaccaria, M., Varnier, M., Piazza, P., Noventa, D., & Ermolao, A. (1999). Blunted growth hormone response to maximal exercise in middle-aged versus young subjects and no effect of endurance training. *Journal of Clinical Endocrinology and Metabolism*, 84(7), 2303–2307.

Part VII
Time Course of Anger: State, Trait and the
Persistence of Vengefulness

Chapter 22

The Temporal Dynamics of Anger: Phenomena, Processes, and Perplexities

Michael Potegal

... some emotions may have particular, built-in time courses... (that cannot) be stopped at will... once overt expression has been allowed to go beyond a certain point. Anger need not be very intense to have this sort of inertia... (Frijda, Mesquita, Sonnemans, & Van Goozen, 1991, p. 200)

Abstract Ordinary, everyday “episodes” of anger typically last less than half an hour, but their duration generally increases with intensity. Anger intensity and duration decline with increasing socioeconomic status; intensity, but not duration, declines with age. Anger at home is more intense but shorter than anger at work. Homicidal fantasies and anger-intensifying rumination on the unjust causes of one’s anger occur more frequently in men than women, are experienced as not entirely volitional, and can transform and extend the experience of anger to days, weeks, and months. In the face of constant or repeated provocation, anger escalates in a highly nonlinear fashion, significantly increasing the probability of aggressive action. At high intensities, some people (probably women more than men) experience their anger as out of control. Escalation may involve various forms of positive physiological and behavioral feedback; the feelings associated with it might have some neurological basis in a shift among the frontal cortical areas momentarily controlling behavior.

Within an episode, anger rises and falls; the rise is typically much faster than the fall. The nature of this trajectory, the conditions that affect it (including the possible automatization of anger in repeated conflicts), and its implications for appraisal models of emotion and social information processing models of aggression remain to be explored. Anger can “decay” by itself or can be “quenched” by extrinsic processes such as apology. Catharsis, the supposed “quenching” of anger by self-initiated aggressive action, is largely a misinterpretation of several associated psychological and physiological effects, but there are several seemingly catharsis-like phenomena in both normal and pathological individuals that warrant further investigation.

22.1 Introduction

Suppose that the defendant has freely admitted that he discovered his spouse in an illicit amorous embrace, *in flagrante delicto*. Enraged (he says), he went in search of his pistol, then shot and killed

M. Potegal (✉)
University of Minnesota, 55455, Minneapolis, MN, USA
e-mail: poteg001@umn.edu

the miscreants. At trial, he pleads a “heat-of-passion” defense, which is still recognized and frequently used, in the United Kingdom, Canada, and all 50 states in the United States. Much is at stake. A successful plea will transform a charge of murder into a verdict of voluntary manslaughter and result in a much lighter sentence. The criteria for a heat-of-passion defense include a provocation sufficient to cause a “reasonable person” to lose self-control and to act on impulse and without reflection. The defendant’s testimony regarding the timing of these events is key. Notably, the criteria also specify that the time elapsing between the provocation and the homicide was not enough for a “reasonable person” to have cooled off and that the defendant had not, in fact, cooled off prior to shooting. However, as noted by Averill (1982) in describing this situation, there are no legal guidelines with regard to cooling time and it is up to the jury to decide how long a “reasonable man” might stay angry. In recent years, the provocation defense has been successfully invoked in cases where the accused killed the victim hours (Wyoming v. Jahnke, 1984; Canada v Thibert, 1996), days (Texas v Watkins, 1999), or weeks (Connecticut v. Martinez, 1991) after the provocation.

Were the judges and the juries right? When someone gets angry, how long do they stay angry? As we will see, if the defendant took more than 20–30 min to find his pistol, the heat-of-passion defense might not apply (although a half hour is about the upper limit for the duration of everyday anger, the more intense anger generated by spousal infidelity might last longer). The legal situation is further confounded by the willingness of some judges to allow the provocation defense to apply to cases of “slow burn” (Bandalli, 1995; Gough, 1999). The nature and time course of a “slow burn” has not been formally defined or analyzed, but Wexler (1999, p. 132) implies that it is characteristic of Type II (“family only”) batterers, dependent and jealous men who “. . . tend to suppress emotions and withdraw, later erupting into violence after long periods of unexpressed but seething rage.” These “long periods” presumably refer to days, weeks, months, or longer. (Note, by the way, that “slow burn” and “cool down” are extensions of the metaphor of anger as heat [Chapter 10 by Koveces, this book]). Wexler’s observation also raises the question of what legal provision should be made for the “unreasonable” man or woman? Linehan (1987) suggests, e.g., that individuals with borderline personality disorder (BPD) routinely take longer to cool down following a provocation (other peculiarities in the time course of BPD anger are noted below).

The importance of understanding the dynamics of anger goes beyond the complexities and contradictions of provocation law. Anger is an internal state that typically outlasts the event(s) that trigger it. What determines such persistence? In a given episode, anger rises and falls. What are the characteristics of this temporal trajectory? Some data reviewed below suggest a rapid rise and a slow decline. What intrapersonal, interpersonal, and situational processes control this trajectory? Appraisal processes are now a standard feature of emotion theories (see Chapter 15 by Wranik and Scherer and Chapter 16 by Berkowitz, this volume, for a respective review and critique of appraisal models). However, the historical assumption that appraisals are almost instantaneous (e.g., Lazarus & Folkman, 1984) was not based on empirical observation, and these models are just beginning to make contact with the real time course of observable phenomena (e.g., Lanctôt & Hess, 2007; see also Chapter 15 by Wranik and Scherer, this book). Consistent with older ideas, the very rapid rise of anger places significant constraints on the depth of processing, but the question of if and how appraisals may contribute to the time course of anger is not just unanswered, it is unasked. Arguably, the rising phase of anger, if not its entire trajectory, represents the envelope of appraisal processes for which these models should account. Social information processing (SIP) models of aggression are the equivalent of appraisal models of emotion in proposing sequential steps from the perception of threat to aggressive action (de Castro, Merk, Koops, Veerman, & Bosch, 2005). SIP models of aggression have focused on cognitive decision-making, largely neglecting the role of emotional processes, but SIP researchers have acknowledged that rapidly rising anger can cause processing steps to be skipped and aggression to be initiated quickly (de Castro et al., 2005).

One of the most salient characteristics of anger is that it can escalate even when the provocation remains constant. It is the escalation of anger that often leads to aggressive acts in children (e.g., Snyder, Edwards, McGraw, Kilgore, & Holton, 1994) and adults (e.g., Felson, 1984). As captured in the idea of a “slow burn” or in the “straw that broke the camel’s back” Arab folktale, this involves a string of relatively mild frustrations or provocations whose accumulating effect eventually produces an abrupt, out-of-control explosion of anger and aggression. This nonlinear “last straw” effect has been invoked in the context of child abuse (Knutson & Bower, 1994), interpersonal conflict (Baumeister, Stillwell, & Wotman, 1990), domestic and criminal violence (Robins & Novaco, 1999; Tsytarev & Grodnitzky, 1995; Wexler, 1999), and suicide (Jenkins & Singh, 2000).

In general, any theory of anger must provide an account of temporal dynamics if it is to be considered complete. As a challenge to future research and theory construction, this chapter first reviews what is known about the parameters and mechanisms governing the duration of anger, then covers aspects of its rise, overall trajectory, and fall. This discussion is informed by the author’s experience with children’s tantrums, but includes the adult experience of anger as well.

22.2 The Persistence of Anger

There are at least two distinct views of the duration of emotions in general and anger in particular. Investigators focusing primarily on the acute, response aspects of emotion, e.g., facial expressions, have suggested that they generally last for a few seconds at most (e.g., Levenson, 1988). Events of this duration are seen in daily life, e.g., flashes of anger lasting no more than a few seconds occur during parent–child interactions (Snyder, Stoolmiller, Wilson, & Yamamoto, 2003). However, much longer episodes of anger also occur in daily life (e.g., Scherer & Wallbott, 1994). Gross (1998) refers to the shorter events as *emotions*, and to the longer ones, which can include more extended social interaction, as *emotion episodes* (cf. state anger, Chapter 23 by Spielberg and Reheiser, this book). Developmentally, according to parent report, the mean duration of tantrums increases from about 2 min at 18 months to around 4 and 5 min in 3- and 4-year-olds, respectively (Potegal, Davidson, 2003). Excessively prolonged anger in children can have clinical significance. Externalizing (e.g., hyperactive, oppositional) 5-year-olds have longer durations, but not higher frequencies, of angry distress when interacting with their mothers in a laboratory frustration task (Cole, Teti, & Zahn-Waxler, 2003). In the more naturalistic home situation, we have found that the tantrums of both externalizing and internalizing (e.g., sad, anxious) 4-year-olds are significantly longer than those of typically developing children (Potegal, 2005).

For adults, at least six psychological surveys of Russian, Japanese, and American subjects consistently found a relatively short mode of typical anger duration varying from “a few minutes” to 5–30 min (e.g., Kassinove, Sukhodolsky, Tsytarev, & Solovyova, 1997; see Potegal, Kosorok, & Davidson, 1996, for review of earlier studies). The many “excitation transfer” studies that successfully demonstrated that the angry arousal provoked in one experimental situation would transfer to another all used intervals of around 5 min, in keeping with the psychological survey results (see Miller, Pedersen, Earlywine & Pollock, 2003, for review). Various acute and chronic individual and situational factors can influence duration. For example, anger directed at the self is reportedly shorter and less intense than anger directed at others (it also occurs less frequently, Chapter 15 by Wranik and Scherer, this book). As might be expected, duration increases with anger intensity in children (Potegal et al., 2003) and adults (Gates, 1926, Table VI; Fridhandler & Averill, 1982; Mikula, Scherer, & Athenstaedt, 1998; Stets & Tsushima, 2001, $r=.35$). In contrast to the psychological surveys, mean reported durations of anger in the sociological 1996 General Social Survey

(GSS) were in the range of hours (Simon & Nath, 2004; Stets & Tsushima, 2001). In this survey, women reported slightly but significantly longer episodes of anger than did men. These data also indicated that higher SES was associated with shorter and less intense anger (Stets & Tsushima, 2001). In contrast, Schieman (2000) found a negatively accelerated increase of anger duration (but a reduction in its overt display) with years of education. Although significant, these effects tended to be small ($0.18 < r < 0.22$). The intensity–duration correlation did not seem to hold with regard to the locus of anger, which was reportedly more intense but shorter at home than at work (Stets & Tsushima, 2001). Similarly, increasing age is associated with a reduction in the frequency and intensity of anger, but not in its duration (Stets & Tsushima, 2001; Chapter 19 by Schieman, this book). There are also cultural differences with Japanese experiences of anger reported to be shorter and less intense than those of Americans (Matsumoto, Kudoh, Scherer, & Wallbott, 1988).

Some of the surveys also found a second mode of duration which lasted hours or days (e.g., Kassinove et al., 1997). This prolonged mode, the “I’m *still* angry about it” reaction, probably does not reflect the original trajectory of anger. More likely, it indicates either the re-activation of anger when subjects were asked to recall an anger-provoking incident and/or ongoing active rumination about the incident. According to several self-report studies, aggressive fantasies and rumination (“brooding”) are a common delayed response to a frustrating or threatening experience (e.g., Caprara, Perugini, Barabaranelli, 1994; Crabb, 2000; Kenrick & Sheets, 1993; Sukhodolsky, Golub, & Cromwell, 2001; Chapter 19 by Tripp and Bies, this book). Like actual physical aggression, such fantasies are more frequent and prolonged among men (60–79% report having them) than among women (32–66% report them, Crabb, 2000; Kenrick & Sheets, 1993). Among those reporting such fantasies, 52% of men and 37% of women reported that their most recent one lasted “more than a few minutes” (i.e., hours, days, or longer, Kenrick & Sheets, 1993). Similarly, the anger of consumers who were contemplating revenge for service failures had a half-life of more than a couple weeks (see Chapter 24 by Tripp and Bies, this volume). At a greater extreme, women who had been divorced reported elevated levels of state anger when interviewed at least 4 years later (mean elapsed time between divorce and interview was 8.9 years, Dreman, Spielberger, & Darzi, 1997). The contribution to this effect of the greater economic burdens and the lower quality of life that typically follows divorce were not investigated.

There is experimental confirmation that rumination prolongs and intensifies anger. Subjects in Rusting and Nolen-Hoeksema’s (1998) widely cited study were presented with an anger-inducing story and instructed to imagine that its unjust events were happening to them. The rumination groups were then asked to “think about” a set of items that were self-focused on emotion (e.g., “why people treat you the way they do”), while the distraction groups were asked to focus attention on external non-emotional items (e.g., think about “the layout of the local post office”). When assessed over a 5–8 min period after the anger induction, the responses of the rumination groups on mood questionnaires and story completion tasks indicated higher levels of anger than the distraction group. The period over which rumination has been experimentally demonstrated to operate was subsequently extended by Bushman, Bonacci, Pedersen, Vasquez, and Miller (2005). In their study, students who were subjected to a demeaning evaluation of their work and then complied with the Rusting and Nolen-Hoeksema (1998) rumination instructions were more aggressive in responding to a mild criticism than did students who had not been instructed to ruminate when both groups were tested 8 h later.

Naturally occurring rumination is reported to be “unintentional” and recurrent; it tends to increase dysphoric mood and interfere with the ability to solve interpersonal problems (Sukhodolsky et al., 2001). As described in Chapter 7 by Stemmler, this volume, a transient rise in blood pressure (BP) is one of the physiological characteristics of anger. Experimentally induced rumination delays the return of BP to baseline in an acute anger recall task (e.g., Gerin, Davidson, Christenfeld, Goyal, &

Schwartz, 2006) and, when anger is chronically suppressed, also tends to reduce the beneficial effect of adaptive self-assertion on BP as well as undercut the beneficial effect of support seeking, at least for men (Hogan & Linden, 2004).

Self-reported aggressive fantasies often involve both weapons and complex strategies (Kenrick & Sheets, 1993), indicating that cognitive neo-associationistic networks are activated during angry rumination (Chapter 16 by Berkowitz, this book). Hedonic quality may promote rumination; Chapter 17 by Litvak et al. (this book) qualify Sukhodolsky et al.'s (2001) characterization of rumination as dysphoric by noting that while looking back at anger experiences can be distressing, looking forward to acting on anger-inspired revenge is pleasant. Sukhodolsky et al. (2001) argue that rumination involves three processes. The first two are memories of past anger episodes, which can trigger new episodes of state anger, and attention to the anger experience, which can amplify its intensity and extend its duration. These effects are probably mediated by the tendency to focus on the causes of anger, typically the injustices perpetrated by the offender (Rusting & Nolen-Hoeksema, 1998). Sukhodolsky et al.'s (2001) third process was "counterfactual" thoughts that may relate to action tendencies toward regret, resolution, or retaliation ("I should have done. . ."). As described in Chapter 24 by Tripp and Bies (this book), perceived injustice is a major cause of anger in the workplace, which then leads to thoughts (and sometimes plans and acts) of revenge. Although the vengeful act itself may be executed without acute feelings of anger, the angry rumination that builds up to it can persist for days, even weeks and months, if not longer. If the (sometimes) legally accepted notion of a "slow burn" has any basis, it might refer to such effects of rumination. In light of the foregoing, it is entirely appropriate that one component of treatment for anger involves training people in "thought-stoppage" techniques for forestalling or terminating rumination (Chapter 28 by Fernandez, this book).

22.3 The Escalation of Anger

As noted above, even when provocation remains constant (e.g., a ceaseless, annoying sound; non-compliance despite repeated requests), or the same provocation is repeated, anger tends to escalate. This ubiquitous phenomenon may account for the sequence of escalating behaviors that recurs in the retrospective self-reports of angry interactions in samples of the "general population," ex-criminal offenders, and ex-mental patients (Felson, 1984, Table 6.3). The reported sequence included "reproaches" (e.g., complaints), "insults" (including yelling), and then threats. Sometimes the sequence then progressed to physical attack. However, these were all interactive events in which it is impossible to disentangle intra-individual vs. inter-individual processes. A similar sequence has been demonstrated under controlled laboratory conditions (Mikolic, Parker, & Pruitt, 1997; Pruitt, Parker, & Mikolic, 1997). In these studies, a stereotyped progression of responses, from mild requests to impatient demands, and then complaints, angry statements, threats and harassment, was observed in people participating in a money-making laboratory task in which the experimenter's confederate consistently withheld needed supplies. This stepwise escalation was shown to form a systematic "hierarchy" of anger responses by Guttman scale analyses (Guttman scaling measures the transitive order of items). Because the confederate's noncompliance remained relatively constant throughout the task, the observed escalation must have been due to changes within the subjects.

That anger escalates in the face of continuing frustration will not come as news to parents who have heard the unmet vocal demands of their children become progressively louder. In our own work (Potegal, Robison, Anderson, Jordan, & Shapiro, 2007), mothers of 15-month-olds prevented their children from playing with a toy by restraining their arms on two consecutive 30 s trials. Within

each trial, physical struggling was the first and most frequent response; children who struggled were significantly more likely to vocalize, and those who vocalized were significantly more likely to show facial expressions of anger. This response hierarchy, too, met criteria for Guttman scalability. The finding that children as young as 15 months show a systematic progression of anger behaviors suggests that such escalation may have a biological basis.

On a functionalist account of emotion, anger is an instrument of interpersonal threat, intimidation, coercion, and domination (e.g., Fridlund, 1991; Lennon & Eisenberg, 1987). As proposed by game theoretic accounts of analogous displays of threat in other animals (e.g., Archer & Huntingford, 1994), evolutionary forces might have shaped these behavior such that individuals spontaneously employ less effortful and lower risk displays of anger before moving on to more effortful and higher risk displays. Distal adaptive ends such as these must be served by proximal psychological mechanism(s). Evidence that increases in self-reported anger are associated with postural (Keltner, Ellsworth, & Edwards, 1993), vocal (Siegman & Snow, 1997), and facial expressions of anger (the “facial feedback” hypothesis, e.g., Rutledge & Hupka, 1985. Adelman & Zajonc, 1989; Hess, Kappas, McHugo, Lanzetta, & Kleck, 1992; Flack, 2006) suggests that short-term escalation may be generated through positive feedback. Feedback from the autonomic activation associated with anger is another possible source (Shields, 1984; Zillmann, 1996; Chapter 7 by Stemmler, this book). In addition to escalation through such “re-afferent” sensory feedback, it is also possible that “corollary discharge” associated with the efferent motor commands to execute the angry expressions and acts serves to increase anger. This latter mechanism has been invoked to explain escalating threat behaviors in other animals (Bond, 1989); demonstrating this sort of mechanism experimentally would be a formidable challenge.

22.4 Anger Priming: A Specific Paradigm of Escalation

Priming refers to the situation in which exposure to a particular stimulus facilitates a response to a subsequent stimulus, as shown by a reduced latency and/or increased frequency and magnitude of responding. Priming occurs in a number of domains (e.g., cognition), but priming in the affective domain may be especially rapid and potent (Murphy & Zajonc, 1993). Many studies have shown that people “primed” by exposure to aggression-related verbal or visual materials are more likely to show an interest in films or other media containing hostility and violence, make hostile attributions about the behavior of others, and engage in aggressive behavior (e.g., Bushman, 1998; Langley, O’Neal, Craig, & Yost, 1992; Todorov & Bargh, 2002). The classical example of such priming is the “weapons effect” first reported by Berkowitz and Le Page (1967), i.e., simply seeing a gun facilitates aggression (e.g., Bartholow, Anderson, Carnagey, & Benjamin, 2005). The priming of anger and aggression, in this sense, is widespread and a socially important phenomenon.

Because the verbal stimuli used in many priming studies require language processing, access to verbal memory, and so forth, these facilitatory effects are likely to involve the fairly general neo-associationistic cognitive networks invoked Chapter 16 by Berkowitz (this book) and others. In contrast, the increased anger that is provoked by repeated exposure to the same frustrating or annoying stimulus is an example of priming that may be more restricted to the specific circuitry of anger. In the two trial arm restraint study with 15-month-olds noted above, the toddler’s responses became notably more probable, rapid, and intense on the second trial. In older children, Chapter 14 by Hubbard et al. (this book) and Chapter 29 by Snyder et al. (this book) report priming of anger in response to successive provocations by a peer and expressions of parental hostility, respectively. Priming by an initial provocation also robustly exacerbates adults’ angry response to a subsequent, more trivial event. Although subject to relatively little direct experimental attention, evidence for this

phenomenon of “triggered displaced aggression” was reportedly found in 82 studies in 50 published articles (Miller et al., 2003). In all cases, the second provocation occurred within 10–20 min of the first, i.e., within the duration of the typical anger episode found by the surveys of anger persistence. As described above, the Bushman et al. (2005) study extended this paradigm to an 8 h delay in which the priming effect was mediated by experimentally induced rumination.

22.5 “Out-of-control” Anger

As noted by Berkowitz, Litvak et al. and others in this book, anger can provide a sense of control to the actor and the perception of control to the onlooker. However, the “unintentional” aspect of rumination noted by Sukhodolsky et al. (2001) suggests that, even at low intensity, anger may be experienced as not completely volitional. Certainly, extreme anger may be associated with a sensed loss of control (Chapter 16 by Berkowitz, Chapter 7 by Stemmler, this book). Chapter 21 by Fessler (this book) argues that such blind rage functions adaptively to drive people into taking risks that will counter or forestall aggression against themselves. Clinically, a perceived loss of control is part of definition of the “anger attacks” that are found with remarkable frequency in unipolar depression (Fava et al., 2000), and possibly also in bipolar depression (Mammen et al., 2004) and seasonal affective disorder (Winkler et al., 2006, for review see Chapter 27 by Novaco, this book). A non-clinical and culturally specific example is the “ataque de nervios” prominent among Caribbean Latinos, but recognized among numerous other Hispanic groups (Guarnaccia et al., 1996). Triggered by serious personal loss (e.g., infidelity, divorce, or death of a loved one), the symptoms of intense anger and sadness include uncontrollable crying and screaming, trembling, and becoming verbally or physically aggressive. Individuals experiencing an ataque de nervios typically report a sense of being out of control (OOC). These experiences are more common among Caribbean Latino women than men, in keeping with the general, cross-national finding that women are more likely to experience anger as a loss of control than are men (e.g., Astin et al., 2003).

OOC seems to capture the subjective essence of an emotion experience as being involuntarily moved from within (e.g., Frijda & Tcherkassof, 1997; Sabini & Silver, 1998); the feeling of OOC in panic attacks may be the most familiar clinical example. Although this phenomenon is rare and difficult to study in adults, some of the behavioral characteristics and governing emotions of ataques de nervios are remarkably similar to the common tantrums of young children. One of the more interesting aspects of tantrums lies in the relatively frequent parental perception of children “losing it,” of becoming OOC. Indicators of being out of control include the inability to be distracted or comforted during the tantrum, the paradoxical result that attempts to distract or comfort may prolong and/or exacerbate the tantrum, and the child’s ambivalence toward or outright refusal of whatever it was that she/he originally wanted. When parents attempt to calm them, the children themselves may say “I can’t stop.”

22.5.1 Mechanisms of OOC

According to Bargh’s (1994) criteria for a typology of cognitive processing, OOC would qualify as an example of *automatic* (vs. *controlled*) processing in that there is a distortion or a loss of the sense of agency (the OOC individual does not experience himself to be the agent of his own behavior). The subjective experience of awareness may also be reduced (the individual is less able to reflect on his own processing). However, individuals in the throes of OOC anger do not meet the “control” criterion in that they are aware that automatic processes might be occurring, although they may feel

unable to counteract them. Barrett, Ochsner, and Gross (2007) argue that controlled processing may be more central to emotion generation than it would seem; even absent any sense of agency or control or intention. Thus, an OOC individual processes what is said (although he may not respond to it), and usually directs anger and aggression to some targets and not others.

Loewenstein (1996, 2000) views the OOC phenomenon from the perspective of economic models of behavior. He argues that when people are under the influence of “visceral factors,” which include emotions such as anger, they make decisions that are inconsistent with a rational calculation of benefit and that “wreak havoc” on their long-term advantage. Visceral factors have these effects by their direct (usually aversive) hedonic impact and also by increasing “the marginal utility of goods and actions that can mitigate the visceral factor.” The heightened preference for these goods and actions increases their “marginal rate of substitution” for other goods and actions and reduces the usual accounting of payoffs for delaying action (“I want it *now*.”) Loewenstein notes that people make these decisions while being aware of acting against their long-term best interest. At lower intensities, the discrepancy between the viscerally driven impulse and the rational calculation of the expected consequences is experienced as a conflict between the urge to do something and the feeling that one should not do it. As the intensity of these factors increase, they focus the individual on the immediate impulse and preclude consideration of other goals, future consequences, and the utility of delaying action. At sufficient intensity, people will sacrifice almost any quantity of other goods for even small amounts of the immediately desired ones. It is when people under the influence of visceral factors make decisions against their long-term self-interest, and are fully aware of doing so, that they feel “out of control.” Although Loewenstein’s main examples are addictive behaviors, he notes that “even at the moment of . . . succumbing to the impetus of road rage” “. . . most people recognize that it is not in their interest to assault a fellow driver who annoys them” (Loewenstein, 2000, p. 428).

Neurochemically, OOC anger attacks in depression are associated with greater serotonergic dysregulation, as measured by a more blunted response to fenfluramine challenge (Fava et al., 2000) and a favorable response to the antidepressants (e.g., Mammen, Pilkonis, Chengappa, & Kupfer, 2004). As noted above, the shift toward automatic processing in OOC may primarily involve a change in the control of action. Neurophysiologically, this could be a switch away from the routine and accustomed control of response choice and initiation by dorsal aspects of lateral and medial frontal cortex and anterior cingulate to the relative novelty of responses initiated and/or monitored by ventrally located structures within or closely allied to the limbic system, such as the orbitofrontal cortex, insula, and amygdala (e.g., Phan et al., 2005). In this context, it is noteworthy that the autonomic feedback from the visceral activation associated with anger is transmitted to these parts of the brain (Berntson, Sarter, & Cacioppo, 2003).

22.6 The Trajectory of Anger

Anger rises, then falls. In Davidson’s (1998) review of “affective chronometry,” he suggests that two key parameters of emotion dynamics are rise time (time from onset to peak) and recovery time (peak to baseline). The sparse data available suggest that the rise of anger is typically quite rapid and the fall considerably slower. This is clearly true in young children’s tantrums, as objectively recorded by their parents (Chapter 12 by Potegal and Qiu, this book), as well as in the on-ward “rages” of older child psychiatry inpatients (Potegal, Carlson, Margulies, Gutkovitch, & Wall, 2009). This effect also appears in adults’ guided reconstruction of remembered experiences (Frijda, Ortony, Sonnemans, & Clore, 1992, Fig. 3.1, p. 68; cf. Tsytarev & Grodnitzky, 1995, Fig. 6.1). Young adults who tracked

their own anger episodes in real time reportedly indicated that the peak of anger occurred at the onset of the episode; the subsequent decline was a slow negative deceleration (Beck & Fernandez, 1998). Chapter 21 by Fessler (this book) quite cogently argues that the suddenness of the “flash” of anger is adaptively significant in being a most effective deterrent of future aggression toward the angry individual. However, anger dynamics can be modified by various factors. In two groups of 3–4-year-olds whose tantrums were recorded with different methods, the longer the overall tantrum duration, the slower was the rise of anger and the later was its peak (Chapter 12 by Potegal and Qiu, this book). If this result is replicable, one possibility among many is that when the initial rate of rise is lower, the anger-inhibitory effect of the distress (sadness) component of tantrums becomes more prominent, thereby delaying and lowering the peak of anger.

Automatization of anger through repeated experience, a learning effect, probably plays a major role in modifying trajectory. Thus, one might expect anger to rise relatively slowly in (the relatively rare) interactions with strangers where appraisal of situation is likely to play a major role (note that this describes the majority of laboratory studies of anger). In contrast, anger is likely to rise quickly in familiar situations, e.g., in family conflicts, which tend to be relatively frequent and well rehearsed. In part, child temperament and adult personality are about individual differences in how rapidly and how high emotion rises and how quickly it resolves. Psychopathology plays a role, too. Vietnam veterans suffering from PTSD experienced a more rapid onset of anger in a relived anger task than did veterans without PTSD (Beckham et al., 2002; Chapter 27 by Novaco, this book).

22.7 The Termination of Anger: Decay, Quenching, Catharsis or . . . ?

However long anger persists, and whatever the vicissitudes of its escalation, it eventually ends. What are the mechanisms by which it does so? To kindle conceptualization and spark research, we compare the processes of “decay,” “quenching,” and “catharsis,” three different physical metaphors for the termination of anger.

22.7.1 *Decay*

The “decay” of anger refers to its gradual decline with the passage of time and without the intrusion of any specific extrinsic stimuli or process. The metaphor of “decay” refers to naturally occurring processes which are somehow intrinsic to the physical system and which show mathematical regularity in the steady diminution of activity or state. Examples of such physical processes might include radioactive decay, leaking of charge from a capacitive circuit, or dissipation of heat from some object. In neural terms, it might reflect activity in specific neural circuitry that gradually diminishes to background levels. Among the very scarce data documenting the termination of anger, decay processes seem to fit the self-reports analyzed by Frijda et al. (1992) and Beck and Fernandez (1998). Waschbusch et al. (2002, Fig. 2 & personal communication) found that after losing to, and being taunted by, an opponent in a computer game, 9–13 years old boys with comorbid diagnoses of ADHD and ODD/CD had significantly slower rates of anger decay over a 1–2 min postgame period than did comparison groups. Methodologically, the many empirical caveats to interpreting anger termination as a decay process include the possibility that a gradual decline that appears in pooled data might mask abrupt and discontinuous changes occurring at different points in the individual data contributing to the pool.

22.7.2 *Quenching*

In contrast to intrinsic decay, quenching refers to the effects of some extrinsic process that overrides, disrupts, or terminates anger. Quenching would be inferred from an abrupt cessation of anger. Consistent with the metaphor of anger as heat (Chapter 10 by Koveces, this book), the trope of quenching anger as one would quench a fire has a deep history in religious and literary writing (e.g., “How mercifully did God quench the fury of the people” Brinklow 1545/1874). Shakespeare’s *Romeo and Juliet* (1597/1992) borrows both trope (“... quench the fire of your pernicious rage...”) and story from an earlier poem by Arthur Brooke (1562/1957).

Anecdotal examples of anger quenching would include temper tantrums interrupted by an interesting event that distracts the child. Among adults, women are more likely to distract themselves when angry than are men, in keeping with gender roles and expectations (Rusting & Nolen-Hoeksema, 1998). However, the effect of distraction in reducing anger is limited and may succeed only when the individual sees a possibility for the resolution of the conflict which triggered the anger (Rusting & Nolen-Hoeksema, 1998). Under some conditions, fear can override anger (e.g., Chapter 16 by Berkowitz, this book). As reviewed by Petrucci (2002), apology by a perpetrator can also substantially reduce victim’s anger and, in juridical context, lead to less punishment, depending on a number of conditions. Such reduction is limited by the “authenticity” of the apology (which typically must involve a timely, face-to-face interaction in which the perpetrator demonstrates sadness or remorse) and diminishes with increasing magnitude of the offense. Even under these conditions, anger reduction by an offered apology is not a simple or automatic stimulus effect, but depends on the victim’s forgiveness (a response effect which may involve, e.g., re-assessment of the offender’s intent, see Chapter 24 by Tripp and Bies and also citations in Chapter 15 by Wranik and Scherer, this book). Apology may also promote the recovery of BP more consistently than it reduces the subjective experience of anger (Anderson, Linden, & Habra, 2006). These various caveats notwithstanding, the quenching of anger by apology is strong enough an effect to be a major focus of interest in law (or at least in federal sentencing guidelines, Petrucci, 2002). In medicine, apology by practitioners for anger-inducing events is an increasingly popular way to foster better patient relations and avoid malpractice suits (e.g., Lazare, 2006).

The foregoing is not to say that fear and apology quench anger in the same way. As suggested by all the caveats noted above, apology is a complex, cognitively mediated social process marking some closure of the incident. Socially, it would be considered inappropriate to remain angry at someone who has apologized, i.e., if the perpetrator does make amends, the victim’s claim on sympathy or restitution is then diminished (Baumeister et al., 1990). Because it involves the processing of highly nuanced communication, apology must be cortically mediated. Fear, in contrast, may have its quenching effect more through subcortical circuitry. Placing fear and apology in the same category is a reminder that reverberating activity in a given neural circuit can be disrupted by impinging stimuli of various sorts.

Note that there can be a real problem in distinguishing quenching from decay if the quenching process is itself slow, gradual, and intrinsic. Sadness and remorse quite commonly follow anger. Functionally, these emotions are appropriate to restoring valued social bonds ruptured by the anger. In children’s tantrums, at least, sadness overlaps considerably with anger and, as noted above, may operate to slowly inhibit the anger. Other internal inhibitory processes may also quench anger.

22.7.3 *Catharsis*

Catharsis has had a multitude of historical interpretations, but current versions are inspired by Breuer and Freud’s (1893/1974) psychodynamic notion that an “abreaction” of previously unexpressed

emotion related to some trauma is required to eliminate symptoms associated with that trauma. As applied to anger and aggression by social psychologists during the middle decades of the last century, the catharsis hypothesis was a metaphor based on the conservation of energy. Once provoked, anger must be expressed in one form or another. The expression of anger reduces the likelihood of further angry and aggressive action and relieves psychological and physiological tension. (In the current context, catharsis would amount to the special case of “quenching” of anger by self-initiated aggressive action). Unexpressed, “pent up” anger might induce psychopathology, depending on the chronicity and intensity of the frustration and/or provocation, and, in the psychodynamic view, if mediated by associated intrapsychic conflicts.

Because the idea of catharsis caught the popular imagination and because it has inspired advocacy of allegedly therapeutic “venting” (e.g., hitting inanimate objects, Lee, 1993), it has been the focus of extensive research. Theoretically, the proposition that catharsis could occur “vicariously” (e.g., by seeing the wrongdoer punished by a third party) or through aggressive fantasy (Feshbach, 1984) removed this hypothesis from the physical realm and revealed its metaphoric character. Empirically, the research found very little support for the primary hypothesis that the expression of anger reduced aggressive behavior; witnessing or expressing anger generally leads to more aggression, in both the short and long term (for review see Geen & Quanty, 1977; Bushman, 2002). Expressing anger does facilitate a more rapid return of BP to normotensive levels, but so does engaging in a number of other coping responses, including expressing a friendly response to the individual who provoked the anger (Hokanson, Willers, & Koropsak, 1968). Anger induces an impulse to aggressive action, but, in many cases, successful, self-assertive coping with a frustrating or provocative challenge can satisfy the impulse and restore the self-esteem that was damaged by the insult (Averill, 1982).

Within academic psychology, if not the rest of the world, the catharsis hypothesis has generally been discredited (Bushman, 2002). It is enjoying a minor recrudescence in the developmental literature as the belief in supposedly self-regulatory “venting” of anger in children (e.g., Denham et al., 2003). Therapeutically, Fernandez (Chapter 28 this book) includes the Gestalt “Empty Chair” form of catharsis as a third stage of a cognitive-behavioral-affective treatment for anger. Overall effect sizes reported for this treatment range from 0.53 to 0.99.

Whether or not developmental self-regulation research and/or psychotherapy is recapitulating or avoiding the earlier errors of catharsis theory, there are some phenomena whose catharsis-like aspects warrant explanation. Developmentally, some parents report that when their cranky and irritable toddler cannot be comforted, she/he will fuss and whine and eventually have a bout of crying or a full-blown tantrum. Afterward, the child calms down and quickly falls asleep. Some exhausted parents will deliberately trigger such crying, knowing that it will hasten sleep. It may be this sort of effect that has moved some to attribute cathartic effects to tantrums (e.g., Solter, 1998). The adult version of this experience is that some people, especially women, report feeling better after a “good cry.” Survey research and experimental studies have yielded inconsistent results concerning the beneficial effects of crying, with more negative than positive outcomes (Cornelius, 1997). However, indications that crying can reduce tension have emerged from focused retrospective self-reports when shorter rather than longer bouts of weeping are involved (about 20 min vs. about 50 min) and when the weeping actually affected the other individual(s) in the relationship problem at issue (Cornelius, 1997). Similarly, although previous physiological studies have suggested an increase, not a reduction, of sympathetic arousal *during* crying in adults, there is at least one recent report of a counterbalancing rebound of vagal activity (as measured by respiratory sinus arrhythmia) *after* a bout of crying (Rottenberg, Wilhelm, Gross, & Gotlib, 2003).

There are also striking examples of seemingly catharsis-like effects associated with some types of psychiatric and neurological pathology. Among the psychiatric phenomena is the “catathymic crisis” first described by Maier (1912) and brought to wider attention by Wertham (1937) in an attempt to explain seemingly inexplicable acts of aggression by certain individuals against people they had

known for a long time (for review see Schlesinger, 1996; cf. Chapter 27 by Novaco, this book). These cases involve some real or imagined provocation that prompts obsessive rumination. Over the course of weeks or months, the thoughts of these individuals become progressively more egocentric and disturbed. At some point, they decide that a violent act is the only resolution. They struggle against the urge, but eventually attempt to, or actually commit, the act. Following the assault, they experience a relief of tension and a return to apparent normality. Coid (1993) described a quite similar phenomenon occurring over 24–48 h in patients with borderline personality disorder, although he did not use the term catathymic crisis. Revitch and Schlesinger (1981) argued for an even more rapid, “acute” form of catathymic crisis that could involve aggression against a stranger. Overall, the catathymic crisis appears to be a pathological intensification of normal aggressive fantasies and ruminative processes in individuals who may have a history of psychiatric disturbance prior to their crisis and a disinhibition of their aggressive impulses. The relief that follows their aggressive action is a similarly intensified version of the more typical reduction of tension brought about by a successful coping response.

Similar effects appear in a few cases of temporal lobe epilepsy (TLE). For example, Mark and Ervin (1970, pp. 93–94) describe a patient who would brood for hours over an imagined insult, working himself up to rage which would last 5 or 6 min during which he would assault his wife or children. He would then experience remorse or grief, sobbing as uncontrollably as he had raged. Following that, he would fall asleep, then wake up feeling refreshed and eager to work. Remarkably, these patients may feel relaxed, at peace, or even euphoric after a rage seizure. The details of sequence and time course are quite similar across a number of patients (cf. Smith, 1980). A similar time course, involving post-rage euphoria and an “anergic” state, has been described in Intermittent Explosive Disorder (McElroy, Soutullo, Beckman, Taylor, & Keck, 1998). Potegal (1994) has conjectured that the rage may involve activation of the medial amygdala while its cessation involves a rebound inhibition by the lateral amygdala. The preceding discussion is not intended as a brief for the catharsis hypothesis; it is meant to draw attention to some interesting phenomena that warrant a more complete explanation.

22.8 Summary

What have we learned? Ordinary, everyday “episodes” of anger typically last less than half an hour, but their duration generally increases with intensity. In sociological surveys, women’s self-reported anger lasts slightly longer than men’s. According to these surveys, anger intensity and duration decline with increasing socioeconomic status; intensity, but not duration, declines with age. Anger at home is more intense but shorter than anger at work. Homicidal fantasies and anger-intensifying rumination on the unjust causes of one’s anger occur with some frequency (more in men than women), are experienced as not entirely volitional, and can transform and extend the experience of anger to days, weeks, and months. In the face of constant or repeated provocation, anger escalates in a highly nonlinear fashion, significantly increasing the probability of aggressive action. Various forms of anger and aggression “priming” are examples of escalation. At high intensities, some people (probably women more than men) experience their anger as out of control. Escalation may involve various forms of positive physiological and behavioral feedback; the feelings associated with it might have some neurological basis in a shift among the frontal cortical areas momentarily controlling behavior.

Within an episode, anger rises and falls; the rise is typically much faster than the fall. The nature of this trajectory, the conditions that affect it (including the possible automatization of anger in repeated

conflicts), and its implications for appraisal models of emotion and social information processing models of aggression remain to be explored. Anger can “decay” by itself or can be “quenched” by extrinsic processes such as apology. Catharsis, the supposed “quenching” of anger by self-initiated aggressive action, is largely a misinterpretation of several associated psychological and physiological effects, but there are several seemingly catharsis-like phenomena in both normal and pathological individuals that warrant further investigation.

Returning to our starting point, the complexities of anger and its time course render the legal heat-of-passion defense even more problematic than has been appreciated. More generally, this chapter has drawn from disparate fields in assembling and ordering phenomena, both well known and obscure, that reflect the time course of anger. This comparison of similarities and differences is intended to stimulate inquiry into the mechanisms underlying these events. If the attempt to impose order on this material provokes academic pique sufficient to motivate research to overcome any conceptual confusion, it will have done its job.

References

- Anderson, J. C., Linden, W., & Habra, M. E. (2006). Influence of apologies and trait hostility on recovery from anger. *Journal of Behavioral Medicine*, 29, 347–358.
- Archer, J., & Huntingford, F. (1994). Game theory models and escalation of animal fights. In M. Potegal & J. F. Knutson (Eds.), *The dynamics of aggression: Biological and social processes in dyads and groups* (pp. 3–31). Hillsdale, NJ: Lawrence Erlbaum Associates.
- Astin, S., Redston, P., & Campbell, A. (2003). Sex differences in social representations of aggression: Men justify, women excuse? *Aggressive Behavior*, 29, 128–133.
- Averill, J. R. (1982). *Anger and aggression*. New York: Springer Verlag.
- Bandalli, S. (1995). Provocation: A cautionary note. *Journal of Law and Society*, 22, 398–409.
- Bargh, J. A. (1994). The four horsemen of automaticity: Awareness, intention, efficiency, and control in social cognition. In R. S. J. Wyer, & T. K. Srull (Eds.), *Handbook of social cognition*, vol. 1: Basic processes; vol. 2: Applications (2nd ed., pp. 1–40). Hillsdale, NJ: Lawrence Erlbaum Associates, Inc.
- Barrett, L. F., Ochsner, K. N., & Gross, J. J. (2007). On the automaticity of emotion. In J. Bargh (Ed.), *Social psychology and the unconscious: The automaticity of higher mental processes* (pp. 173–217). New York: Psychology Press.
- Bartholow, B. D., Anderson, C. A., Carnagey, N. L., & Benjamin, A. J., Jr. (2005). Interactive effects of life experience and situational cues on aggression: The weapons priming effect in hunters and nonhunters. *Journal of Experimental Social Psychology*, 41, 48–60.
- Baumeister, R. E., Stillwell, A., & Wotman, S. R. (1990). Victim and perpetrator accounts of interpersonal conflict: Autobiographical narratives about anger. *Journal of Personality and Social Psychology*, 59, 994–1005.
- Beck, E., & Fernandez, R. (1998). Cognitive-behavioral self-regulation of the frequency, duration, and intensity of anger. *Journal of Psychopathology and Behavioral Assessment*, 20, 217–229.
- Beckham, J. C., Vrana, S. R., Barefoot, J. C., Feldman, M. E., Fairbank, J., & Moore, S. D. (2002). Magnitude and duration of cardiovascular responses to anger in Vietnam veterans with and without posttraumatic stress disorder. *Journal of Consulting and Clinical Psychology*, 70, 228–234.
- Berkowitz, L., & Le Page, A. (1967). Weapons as aggression-eliciting stimuli. *Journal of Personality and Social Psychology*, 7, 202–207.
- Berntson, M., Sarter, J. T., & Cacioppo, G. G. (2003). Ascending visceral regulation of cortical affective information processing. *European Journal of Neuroscience*, 18, 2103–2109.
- Bond, A. B. (1989). Toward a resolution of the paradox of aggressive displays. II: Behavioral efference and the communication of intentions. *Ethology*, 81, 235–249.
- Breuer, J., & Freud, S. (1974). On the psychical mechanism of hysterical phenomena: Preliminary communication. In J. Breuer & S. Freud (Eds.), *Studies on hysteria* (J. Strachey & A. Strachey, Trans.) (pp. 53–69). Harmondsworth and New York: Penguin Books. (Original publication 1893)
- Brinklow, H. (c. 1545/1874). Complaynt of Roderick Mors. In J. M. Cowper (Ed.), *The lamentacion of a Christian against the cite of London, made by Roderigo Mors*. London: Published for the Early English Text Society by N. Trübner & Co.

- Brooke, A. (1957). The tragi-call historye of Romeus and Juliet written first in Italian by Bandell, and now in English. In B. Geoffrey (Ed.), *Narrative and dramatic sources of Shakespeare*. London and New York: Columbia University Press.
- Bushman, B. J. (1998). Priming effects of media violence on the accessibility of aggression constructs in memory. *Personality & Social Psychology Bulletin*, 24, 537–545.
- Bushman, B. J. (2002). Does venting anger feed or extinguish the flame? Catharsis, rumination, distraction, anger, and aggressive responding. *Personality and Social Psychology Bulletin*, 28, 724–731.
- Bushman, B. J., Bonacci, A. M., Pedersen, W. C., Vasquez, E. A., & Miller, N. (2005). Chewing on it can chew you up: Effects of rumination on triggered displaced aggression. *Journal of Personality and Social Psychology*, 88, 969–983.
- Canada v Thibert (1996). 1 S.C.R. 37.
- Caprara, G. V., Perugini, M., & Barabaranelli, C. (1994). Studies of individual differences in aggression. In Potegal, M. and Knutson, J. (Eds.), *The dynamics of aggression: Biological and social processes in dyads and groups* (pp. 123–154). Hillsdale, NJ: Lawrence Erlbaum Associates, Inc.
- Coid, J. W. (1993). An affective syndrome in psychopaths with borderline personality disorder? *British Journal of Psychiatry*, 162, 641–650.
- Cole, P. M., Teti, L. O., & Zahn-Waxler, C. (2003). Mutual emotion regulation and the stability of conduct problems between preschool and early school age. *Development and Psychopathology*, 15, 1–18.
- Connecticut v. Martinez (1991). 591 A.2d 155.
- Cornelius, R. R. (1997). Toward a new understanding of weeping and catharsis? In F. J. Van Bussel & A. J. W. Boelhouwer, (Eds.), *The (non)expression of emotions in health and disease* (pp. 303–321). Tilburg, the Netherlands: Tilburg University Press.
- Crabb, P. B. (2000). The material culture of homicidal fantasies. *Aggressive Behavior*, 26, 225–234.
- Davidson, R. J. (1998). Affective style and affective developer: perspective from affective neuroscience. *Cognition and Emotion* 12, 307–330
- Denham, S. A., Blair, K. A., DeMulder, E., Levitas, J., Sawyer, K., Auerbach-Major, S., et al. (2003). Preschool emotional competence: Pathway to social competence? *Child Development*, 74, 238–256.
- Dreman, S., Spielberger, C., & Darzi, O. (1997). The relation of state-anger to self-esteem, perceptions of family structure and attributions of responsibility for divorce of custodial mothers in the stabilization phase of the divorce process. *Journal of Divorce & Remarriage*, 28, 157–170.
- Fava, M., Vuolo, R. D., Wright, E. C., Nierenberg, A. A., Alpert, J. E., & Rosenbaum, J. F. (2000). Fenfluramine challenge in unipolar depression with and without anger attacks. *Psychiatry Research*, 94, 9–18.
- Felson, R. B. (1984). Patterns of aggressive social interaction. In A. Mummendey (Ed.), *Social psychology of aggression* (pp. 107–126). New York: Springer-Verlag.
- Feshbach, S. (1984). The catharsis hypothesis, aggressive drive, and the reduction of aggression. *Aggressive Behavior*, 10, 91–101.
- Flack, W. F., Jr. (2006). Peripheral feedback effects of facial expressions, bodily postures, and vocal expressions on emotional feelings. *Cognition and Emotion*, 20, 177–195.
- Fridhandler, B. M., & Averill, J. R. (1982). Temporal dimensions of anger: An exploration of time and emotion. In J. R. Averill (Ed.), *Anger and aggression* (pp. 253–280). New York: Springer-Verlag.
- Fridlund, A. J. (1991). Evolution and facial action in reflex, social motive, and paralanguage. *Biological Psychology*, 32, 3–100.
- Frijda, N. H., Mesquita, B., Sonnemans, J., & Van Goozen, S. (1991). The duration of affective phenomena or emotions, sentiments, and passions. In K. T. Strongman (Ed.), *International review of studies of emotion* (Vol. 1, pp. 187–226). Chichester: Wiley & Sons.
- Frijda, N. H., Ortony, A., Sonnemans, J., & Clore, G. (1992). The complexity of intensity. In M. Clark, et al. (Eds.), *Emotion. Review of personality and social psychology* (Vol. 13, pp. 60–89). Thousand Oaks, CA: Sage Publications.
- Frijda, N. H., & Tcherkassof, A. (1997). Facial expressions as modes of action readiness. In J. A. Russell, J. M. Fernandez-Dols, (Eds.), *The psychology of facial expression* (pp. 78–102). Cambridge: Cambridge University Press.
- Gates, G. S. (1926). An observational study of anger. *Journal of Experimental Psychology*, 9, 325–336.
- Geen, R. G., & Quanty, M. B. (1977). The catharsis of aggression: An evaluation of a hypothesis. In Berkowitz, L., (Ed.), *Advances in experimental social psychology* (Vol. 10, pp. 1–37). New York: Academic Press.
- Gerin, W., Davidson, K., Christenfeld, N. J. S., Goyal, T., & Schwartz, J. E. (2006). The role of angry rumination and distraction in blood pressure recovery from emotional arousal. *Psychosomatic medicine*, 68, 64–72.

- Gough, S. (1999). Taking the heat out of provocation. *Oxford Journal of Legal Studies*, 19, 481–494.
- Gross, J. J. (1998). The emerging field of emotion regulation: An integrative review. *Review of General Psychology*, 2, 271–299.
- Guarnaccia, P. J., Rivera, M., Franco, F., & Neighbors, C. (1996). The experiences of *Ataques de Nervos*: Toward an anthropology of emotions in Puerto Rico. *Culture Medicine and Psychiatry*, 20, 343–367.
- Hess, U., Kappas, A., McHugo, G. J., Lanzetta, J. T., & Kleck, R. E. (1992). The facilitative effect of facial expression on the self-generation of emotion. *International Journal of Psychophysiology*, 12, 251–265.
- Hogan, B. E., & Linden, W. (2004). Anger response styles and blood pressure: At least don't ruminate about it! *Annals of Behavioral Medicine*, 27, 38–49.
- Hokanson, J. E., Willers, K. R., & Koropsak, E. (1968). The modification of autonomic responses during aggressive interchange. *Journal of Personality*, 36, 386–404.
- Jenkins, R., & Singh, B. (2000). Policy and practice in suicide prevention. *The British Journal of Forensic Practice*, 2, 3–11.
- Kassinove, H., Sukhodolsky, D., Tytsarev, S. V., & Solovyova, S. (1997). Self-reported anger episodes in Russia and America. *Journal of Social Behavioral Personality*, 12, 301–324.
- Keltner, D., Ellsworth, P. C., & Edwards, K. (1993). Beyond simple pessimism: Effects of sadness and anger on social perception. *Journal of Personality and Social Psychology*, 64, 740–752.
- Kenrick, D. T., & Sheets, V. (1993). Homicidal fantasies. *Ethology and Sociobiology*, 14, 231–246.
- Knutson, J. F., & Bower, M. E. (1994). Physically abusive parenting as an escalated aggressive response. In M. Potegal & J. F. Knutson (Eds.), *The dynamics of aggression: Biological and social processes in dyads and groups* (pp. 3–31). Hillsdale, NJ: Lawrence Erlbaum Associates.
- Langtôt, N., & Hess, U. (2007). The timing of appraisals. *Emotion*, 7, 207–212.
- Langley, T., O'Neal, E. C., Craig, K. M., & Yost, E. A. (1992). Aggression-consistent, -inconsistent, and -irrelevant priming effects on selective exposure to media violence. *Aggressive Behavior*, 18, 349–356.
- Lazare, A. (2006). Apology in medical practice an emerging clinical skill. *JAMA*, 296, 1401–1404.
- Lazarus, R. S., & Folkman, S. (1984). *Stress, appraisal and coping*. New York: McGraw-Hill.
- Lee, J. (1993). *Facing the fire: Experiencing and expressing anger appropriately*. New York: Bantam.
- Lennon, R., & Eisenberg, N. (1987). Emotional displays associated with preschoolers' prosocial behavior. *Child Development*, 58, 992–1000.
- Levenson, R. W. (1988). Emotion and the autonomic nervous system: A prospectus for research on autonomic specificity. In H. L. Wagner (Ed.), *Social psychophysiology and emotion: Theory and clinical applications* (pp. 17–42). Chichester: Wiley.
- Linehan, M. M. (1987). Dialectical behavior therapy for borderline personality disorder: Theory and method. *Bulletin of the Menninger Clinic*, 51, 261–276.
- Loewenstein, G. (1996). Out of control: Visceral influences on behavior. *Organizational Behavior and Human Decision Processes*, 65, 272–292.
- Loewenstein, G. (2000). Emotions in economic theory and economic behavior. *The American Economic Review*, 90, 426–432.
- Maier, H. W. (1912). Katathyme Wahnbildung und Paranoia. *Zeitschrift für die gesamte Neurologie und Psychiatrie*, 13, 555–610.
- Mammen, O. K., Pilkonis, P. A., Chengappa, K. N., & Kupfer, D. J. (2004). Anger attacks in bipolar depression: Predictors and response to citalopram added to mood stabilizers. *Journal of Clinical Psychiatry*, 65, 627–633.
- Mark, V. H., & Ervin, F. R. (1970). *Violence and the brain*. New York: Harper & Row.
- Matsumoto, D., Kudoh, T., Scherer, K., & Wallbott, H. (1988). Antecedents of and reactions to emotions in the United States and Japan. *Journal of Cross-Cultural Psychology*, 19, 267–286.
- McElroy, S. L., Soutullo, C. A., Beckman, D. A., Taylor, P., & Keck, P. E. (1998). DSM-IV intermittent explosive disorder: A report of 27 cases. *Journal of Clinical Psychiatry*, 59, 203–210.
- Mikolic, J. M., Parker, J. C., & Pruitt, D. G. (1997). Escalation in response to persistent annoyance. Groups vs individuals and gender effects. *Journal of Personality and Social Psychology*, 72, 151–163.
- Mikula, G., Scherer, K. R., & Athenstaedt, U. (1998). The role of injustice in the elicitation of differential emotional reactions. *Journal of Personality and Social Psychology*, 74, 769–783.
- Miller, N., Pedersen, W. C., Earlywine, M., & Pollock, V. E. (2003). A theoretical model of triggered displaced aggression. *Personality and Social Psychology Review*, 7, 75–97.
- Murphy, S. T. & Zajonc, R. B. (1993). Affect, cognition and awareness. Affective priming with optimal and suboptimal stimulus Exposures. *Personality social psychology*, 64, 723–739.
- Orobio de Castro, B., Merk, W., Koops, W., Veerman, J. W., & Bosch, J. D. (2005). Emotions in social information processing and their relations with reactive and proactive aggression in referred aggressive boys. *Journal of Clinical Child and Adolescent Psychology*, 34, 105–116.

- Petrucchi, C. J. (2002). Apology in the criminal justice setting: Evidence for including apology as an additional component in the legal system. *Behavioral Sciences and the Law*, 20, 337–362.
- Phan, K. L., Fitzgerald, D. A., Nathan, P. J., Moore, G. J., Uhde, T. W., & Tancer, M. E. (2005). Neural substrates for voluntary suppression of negative affect: A functional magnetic resonance imaging study. *Biological Psychiatry*, 57, 210–219.
- Potegal, M. (1994). Aggressive arousal: The amygdala connection. In M. Potegal & J. F. Knutson (Eds.), *The dynamics of aggression: Biological and social processes in dyads and groups* (pp. 73–111). Hillsdale, NJ: Lawrence Erlbaum Associates.
- Potegal, M. (2005). Tantrums in externalizing, internalizing and typically developing 4 year olds. Society for Research in Child Development, Atlanta GA, Program Book, p. 31.
- Potegal, M., Carlson, G., Margulies, D., Gutkovitch, Z., & Wall, M. (in press). Rages or temper tantrums? The behavioral organization, temporal characteristics, and clinical significance of angry-agitated outbursts in child psychiatry inpatients. *Child Psychiatry and Human Development*, 40, 621–636.
- Potegal, M., & Davidson, R. J. (2003). Temper tantrums in young children: 1. Behavioral composition. *Journal of Developmental and Behavioral Pediatrics*, 24, 140–147.
- Potegal, M., Kosorok, M. R., & Davidson, R. J. (1996). The time course of angry behavior in the temper tantrums of young children. *Annals of the New York Academy of Sciences*, 794, 31–45.
- Potegal, M., Kosorok, M. R., & Davidson, R. (2003). Temper tantrums in young children: 2. Tantrum duration and temporal organization. *Journal of Developmental and Behavioral Pediatrics*, 24, 148–154.
- Potegal, M., Robison, S., Anderson, F., Jordan, C., & Shapiro, E. (2007). Sequence and priming in 15 month olds' reactions to brief arm restraint: Evidence for a hierarchy of anger responses. *Aggressive Behavior*, 33, 1–11.
- Pruitt, D. G., Parker, J. C., & Mikolic, J. M. (1997). Escalation as a reaction to persistent annoyance. *International Journal of Conflict Management*, 8, 252–270.
- Revitch, E., & Schlesinger, L. B. (1981). *Psychopathology of homicide*. Springfield, IL: Charles C Thomas.
- Robins, S., & Novaco, R. W. (1999). Systems conceptualization and treatment of anger. *Journal of Clinical Psychology*, 55, 325–337.
- Rottenberg, J., Wilhelm, F. H., Gross, J. J., & Gotlib, I. H. (2003). Vagal rebound during resolution of tearful crying among depressed and nondepressed individuals. *Psychophysiology*, 40, 1–6.
- Rusting, C. L., & Nolen-Hoeksema, S. (1998). Regulating responses to anger: Effects of rumination and distraction on angry mood. *Journal of Personality and Social Psychology*, 74, 790–803.
- Rutledge, L. L., & Hupka, R. B. (1985). The facial feedback hypothesis: Methodological concerns and new supporting evidence. *Motivation and Emotion*, 9, 219–240.
- Sabini, M., & Silver, J. (1998). The not altogether social construction of emotions: A critique of Harre and Gillett. *Journal for the Theory of Social Behaviour*, 28, 223–236.
- Scherer, K. R., & Wallbott, H. G. (1994). Evidence for universality and cultural variation of differential emotion response patterning. *Journal of Personality and Social Psychology*, 66, 310–328.
- Schieman, S. (2000). Education and the activation, course, and management of anger. *Journal of Health and Social Behavior*, 41, 20–39.
- Schlesinger, L. B. (1996). The Catathymic crisis, 1912–Present: A review and clinical study. *Aggression and Violent Behavior*, 1, 307–316.
- Schwartz, A. R., Gerin, W., Christenfeld, N., Glynn, L., Davidson, K., & Pickering, T. G. (2000). Effects of an anger recall task on post-stress rumination and blood pressure recovery in men and women. *Psychophysiology*, 37(Suppl. 1), S12.
- Shakespeare, W. (1597/1992). *Romeo and Juliet*. New York: Washington Square Press.
- Shields, S. A. (1984). Reports of bodily change in anxiety, sadness, and anger. *Motivation and Emotion*, 8, 1–21.
- Siegmán, A. W., & Snow, S. C. (1997). The outward expression of anger, the inward experience of anger and CVR: The role of vocal expression. *Journal of Behavioral Medicine*, 20, 29–45.
- Simon, R., & Nath, L. E. (2004). Gender and emotion in the United States: Do men and women differ in self-reports of feelings and expressive behaviors? *American Journal of Sociology*, 109, 1137–1176.
- Smith, J. S. (1980). Episodic rage. In M. Girgis & L. G. Kiloh (Eds.), *Limbic epilepsy and the dyscontrol syndrome*. Amsterdam: Elsevier/North Holland Biomedical Press.
- Snyder, J., Edwards, P., McGraw, K., Kilgore, K., & Holton, A. (1994). Escalation and reinforcement in mother-child conflict: Social processes associated with the development of physical aggression. *Development and Psychopathology*, 6, 305–321.
- Snyder, J., Stoolmiller, M., Wilson, M., & Yamamoto, M. (2003). Child anger regulation, parental responses to children's anger displays, and early child antisocial behavior. *Social Development*, 12, 335–360.
- Solter, A. (1988). *Tears and tantrums*. Goleta, CA: Shining Star Press.

- Stets, J. E., & Tsushima, T. M. (2001). Negative emotion and coping responses within identity control theory. *Social Psychology Quarterly*, 64, 283–295.
- Sukhodolsky, D. G., Golub, A., & Cromwell, E. N. (2001). Development and validation of the anger rumination scale. *Personality and Individual Differences*, 31, 689–700.
- Texas v Watkins (1999). 52 S.W.3d 858.
- Todorov, A., & Bargh, J. A. (2000). Automatic sources of aggression. *Aggression and Violent Behavior*, 7, 53–68.
- Tsytarev, S. V., & Grodnitzky, G. R. (1995). Anger and criminality. In H. Kassinove (Ed.), *Anger disorders: Definition, diagnosis, and treatment* (pp. 91–108). Washington, DC: Taylor & Francis.
- Waschbusch, D. A., Pelham, W. E., Jr., Jennings, J. R., Greiner, A. R., Tarter, R. E., & Moss, H. B. (2002). Reactive aggression in boys with disruptive behavior disorders: Behavior, physiology, and affect. *Journal of Abnormal Child Psychology*, 30, 641–656.
- Wertham, F. (1937). The catathymic crisis: A clinical entity. *Archives of Neurology and Psychiatry*, 37, 974–977.
- Wexler, J. D. B. (1999). The broken mirror: A self psychological treatment perspective for relationship violence. *Journal of Psychotherapy Practice and Research*, 8, 129–141.
- Winkler, D., Pjrek, E., Konstantinidis, A., Praschak-Rieder, N., Willeit, M., Stastny, J., et al. (2006). Anger attacks in seasonal affective disorder. *The International Journal of Neuropsychopharmacology*, 9, 215–220.
- Wyoming v. Jahnke (1984). WY 114, Wyo., 692 P.2d 911.
- Zillmann, D. (1996). Sequential dependencies in emotional experience and behavior. In R. D. Kavanaugh, B. Zimmerbegh, & S. Fein (Eds.), *Emotion: Interdisciplinary perspectives*. Hillsdale, NJ: Lawrence Erlbaum Associates.

Chapter 23

The Nature and Measurement of Anger

Charles D. Spielberger and Eric C. Reheiser

Abstract The nature of anger, hostility, and aggression and the relations among these concepts are considered in this chapter. The evolution of the concepts of anger and aggression from the ancient writings of Plato and Aristotle to those of Darwin and Freud is noted. Recent research findings on the relationships between anger, Type A behavior, and coronary heart disease (CHD) are also reviewed, and the essential distinction between anger as an emotional state and individual differences in anger as a personality trait is clarified. Conceptual definitions of anger, hostility, and aggression are examined as components of the AHA! Syndrome, in which anger is clearly an essential emotional motivator of both hostility and aggression. The chapter concludes with a discussion of the ubiquitous nature of anger in daily life and the use of the State–Trait Anger Expression Inventory (STAXI) to assess the experience, expression, and control of anger in research and the diagnosis and treatment of anger-related problems.

Fear and rage were recognized by Darwin (1872/1965) as universal characteristics of both humans and animals that have evolved through a process of natural selection because these emotions facilitated successful adaptation and survival. Darwin observed that both fear and rage were reflected in facial expressions (e.g., reddened face, clenched teeth, dilated nostrils) and were associated with accelerated heart rate, muscular tension, and aggressive, often violent behavior. Tavris (1982, p. 32), in her review of Darwin's observations of rage, concluded that "The face of rage is not learned. It is as much a part of species equipment as a nose or a pair of eyebrows" (cf. Chapter 8 by Matsumoto et al., this book).

Rage was regarded by Darwin (1872/1965, p. 74) as intense anger that motivated "... animals of all kinds, and their progenitors before them, when attacked or threatened by an enemy ..." to fight and defend themselves. For Darwin, anger was a state of mind that differed "... from rage only in degree, and there is no marked distinction in their characteristic signs" (1872/1965, p. 244). Thus, Darwin implicitly defined anger as an emotional state that varies in intensity, from mild irritation or annoyance to intense fury and rage.

The significance of anger as a fundamental emotion and a potential source of human problems was recognized almost 2,500 years ago by Plato, who noted that anger was an extremely negative emotional reaction that should be controlled by means of reason (Tavris, 1982). Aristotle, Seneca, and Plutarch also defined anger as a strong emotion provoked by the perception of being treated badly that motivated a desire for vengeance (DiGiuseppe & Tafrate, 2007). According to

C.D. Spielberger (✉)
University of South Florida, Tampa, FL, USA
e-mail: spielber@cas.usf.edu

Seneca, "... anger clouded a person's judgment, impaired interpersonal effectiveness, and could collectively imbalance an entire society" (DiGiuseppe & Tafrate, 2007, p. 4).

While Darwin viewed anger and aggression as adaptive, Freud emphasized the destructive, violent aspects of aggression (Tavris, 1982). According to Freud's (1933/1959) psychoanalytic theory, aggression was considered a fundamental instinctual drive, consisting of intense angry feelings that motivated aggressive behavior. Aggression, accompanied by anger and rage, was assumed to be motivated by a biologically determined "death instinct" (Thanatos). However, Thanatos was generally inhibited by a more powerful life instinct (libido), which resulted in the energy associated with this self-destructive drive to be expressed in aggressive behavior directed toward other persons or objects in the environment. When aggression could not be directly expressed, anger was turned back into the self, resulting in depression and other psychosomatic manifestations (Alexander & French, 1948; see Chapter 27 by Novaco, this book).

Anger, hostility, and aggression have also been regarded as important factors in essential hypertension and coronary heart disease for a number of years (see Diamond 1982). More than 50 years ago, Franz Alexander theorized that the strenuous efforts of hypertensives to suppress their angry feelings resulted in chronic activation of the cardiovascular system and, eventually, to fixed elevations in blood pressure. Impressive evidence of a strong relationship between suppressed hostility ("anger-in") and hypertension was also reported in research by Harburg and his associates (Gentry, Chesney, Gary, Hall, & Harburg, 1982; Harburg et al., 1973).

Flanders Dunbar (1943), a pioneer in psychosomatic medicine, was among the first to note an association between aggression and coronary heart disease (CHD). She identified a "coronary personality" in CHD patients, whom she described as ambitious, hard-driving, and markedly aggressive, with a strong need for achievement and success. Friedman and Rosenman (1974), in their Western Collaborative Group Study (WCGS), observed similar attitudes and behaviors in CHD patients, which they labeled as Type A Behavior. The Type A Behavior Pattern (TABP) was defined as an action-emotion syndrome characterized by strong ambition, competitiveness, achievement striving, impatience, an extreme sense of time urgency, and emotional responses such as irritation, aggressiveness, and hostility (Friedman & Rosenman, 1974). According to Friedman and Rosenman (1974, p. 59): "Persons possessing this pattern are quite prone to exhibit a free-floating but extraordinarily well-rationalized hostility, which was likely to flare up under very diverse conditions."

In a follow-up study, Matthews, Glass, Rosenman, and Bortner (1977) reviewed and rated the taped interviews of participants in the WCGS study and examined their responses to the interview questions that were considered to reflect Type A or Type B behavior. Subjects' voice modulation, energy level, tendency to interrupt the interviewer, psychological defenses, and potential for hostility were also evaluated. Matthews et al. identified five factors with eigenvalues greater than 1.00: competitive drive, impatience, past achievements, non-job achievement, and speed. However, only competitive drive and impatience were related to heart disease. Further analysis of the competitive drive factor identified "Potential for hostility" as the single item having the strongest significant relationship with CHD ($p < .003$). Individuals who developed CHD were also rated significantly higher than age-matched healthy controls on "Anger directed outward"; "Subject gets angry more than once a week"; and "Competitive in games with peers." All of these characteristics were either directly related to anger/hostility or possibly motivated by anger. Thus, anger and hostility would seem to be major coronary-prone components of the TABP.

The results of recent studies of the association of hostility with coronary artery disease (CAD) provide further evidence of a significant relationship between anger/hostility and CHD. Williams, Barefoot, and Shekelle (1985) found that hostility and cynicism were related to the presence and severity of CAD, as measured by coronary angiography. Similarly, Dembroski, MacDougall, Williams, Haney, and Blumenthal (1985) reported that potential for hostility was associated with

CAD, but primarily for patients who suppressed their anger (anger-in). These findings highlight the importance of assessing the suppression and control of anger/hostility, in addition to measuring how often angry feelings are experienced and expressed. See Chapter 25 by Williams, this book, for a more extended review of these issues.

This chapter examines conceptual definitions of anger, hostility, and aggression and briefly evaluates several instruments developed to assess these constructs. The essential distinction between anger as an emotional state and as a personality trait and the construction and validation of the *State–Trait Anger Scale* (STAS) are also reviewed. The chapter concludes with a brief discussion of the assessment of the experience, expression, and control of anger with the *State–Trait Anger Expression Inventory* (STAXI).

23.1 Anger, Hostility, and Aggression

The maladaptive effects of anger are traditionally emphasized as important contributors to the etiology of psychoneuroses, depression, and schizophrenia. While much has been written about the negative impact of anger and hostility on physical health and psychological well-being, the definitions of these constructs are ambiguous and sometimes contradictory. Moreover, the terms anger, hostility, and aggression are often used interchangeably in the research literature, resulting in conceptual confusion and a diversity of measurement operations of questionable validity (Biaggio, Supplee, & Curtis, 1981).

Anger is defined in the English language in rather diverse terms as this concept has evolved over time. Dictionary definitions have consistently recognized anger as a normal emotional state that varies in intensity and generally occurs as a reaction to being treated badly, unfairly, or abused (see Table 23.1). The words most commonly associated with anger include annoyance, indignation, wrath, fury, and rage, indicating that anger varies in intensity. In essence, anger refers to a psychobiological emotional state or condition that varies in intensity and fluctuates over time (S-Anger). See Chapter 12 by Potegal and Qiu and Chapter 22 by Potegal, this book, for extensions of these ideas .

Table 23.1 Dictionary definitions of anger

Easton's 1897 Bible Dictionary

The emotion of instant displeasure on account of something evil that presents itself to our view. In itself it is an original susceptibility of our nature, just as love is, and is not necessarily sinful. It may, however, become sinful when causeless, or excessive, or protracted. (Matt. 5:22; Eph. 4:26; col. 3:8)

Webster's Dictionary (1913)

A strong passion or emotion of displeasure or antagonism, excited by a real or supposed injury or insult to one's self or others, or by the intent to do such injury. Anger is a feeling of keen displeasure (usually with a desire to punish) for what we regard as wrong toward ourselves or others. It may be excessive or misplaced, but is not necessarily criminal. (p. 56)

Cambridge Advanced Learner's Dictionary (2006)

A strong feeling which makes you want to hurt someone or be unpleasant because of something unfair or hurtful that has happened.

Given the substantial overlap in prevailing conceptual definitions of anger, hostility, and aggression, we have referred collectively to these constructs, as the AHA! Syndrome, and have proposed the following working definitions:

The concept of anger usually refers to an emotional state that consists of feelings that vary in intensity, from mild irritation or annoyance to intense fury and rage. Although hostility usually involves angry feelings, this concept has the connotation of a complex set of attitudes that motivate aggressive behaviors directed toward destroying objects or injuring other people While anger and hostility refer to feelings and attitudes, the concept of aggression generally implies destructive or punitive behavior directed towards other persons or objects (Spielberger, Jacobs, Russell, & Crane, 1983; p. 16).

Anger is clearly at the core of the AHA! Syndrome, and different aspects of this emotion are typically emphasized in various definitions of hostility and aggression, as can be noted in Table 23.2. However, ambiguity and inconsistency in the definitions of anger, hostility, and aggression continue to be reflected in the procedures that have been developed to assess these constructs.

Table 23.2 Definitions of anger, hostility, and aggression

The AHA! syndrome
<i>Anger:</i> Generally refers to an emotional state that consists of feelings that vary in intensity from mild irritation or annoyance to intense fury or rage.
<i>Hostility:</i> Usually involves angry feelings but also has the connotation of a complex set of attitudes that motivate injuring people or damaging objects.
<i>Aggression:</i> Generally refers to destructive or punitive behavior directed toward other persons or objects in the environment.
<i>Hostile Aggression:</i> Aggressive behavior that is motivated by angry feelings.
<i>Instrumental Aggression:</i> Aggressive behavior directed toward a person or obstacle that is not motivated by angry feelings.

23.1.1 Measures of Hostility and Anger

The earliest efforts to assess anger and hostility were based on clinical interviews, behavioral observations, and projective techniques, such as the Rorschach Inkblots and the Thematic Apperception Test. Physiological and behavioral correlates of anger and hostility and various manifestations of aggression have also been investigated in numerous studies. In contrast, the phenomenology of anger, i.e., the experience of angry feelings, has been largely neglected in psychological research. Moreover, most psychometric measures of anger and hostility confound angry feelings with the mode and direction of the expression of anger.

Beginning in the 1950s, a number of self-report psychometric scales were constructed to measure hostility (e.g., Buss & Durkee, 1957; Cook & Medley, 1954; Schultz, 1954). A rational–empirical strategy was employed in developing the Buss–Durkee (1957) Hostility Inventory (BDHI), which is generally regarded as the most carefully constructed early psychometric measure of hostility. Conceptualizing hostility as multidimensional, Buss (1961) developed items to assess seven facets of this construct, each defined by a BDHI subscale. The dimensionality of the BDHI was investigated in two studies in which responses to the individual BDHI items were factored. In contrast to the seven hostility dimensions presumed to be assessed by BDHI subscales, Bendig (1962) found only two underlying factors, which he described as overt and covert hostility. Russell (1981) identified three

meaningful BDHI factors, which were labeled as follows: (1) Neuroticism, (2) General Hostility, and (3) Expression of Anger.

The importance of distinguishing between anger and hostility was explicitly recognized in the early 1970s, marked by the appearance in the psychological literature of three anger measures: The Reaction Inventory (RI), Anger Inventory (AI), and Anger Self-Report (ASR). The RI was developed by Evans and Stangeland (1971) to assess the degree to which anger was evoked by specific situations (e.g., "People pushing into line"). Similar in conception and format, Novaco's (1975) AI inquires about reactions to anger-provoking incidents ("Being called a liar" and "Someone spits at you"). In responding to the RI and the AI, examinees rate the degree to which specific situations or incidents make them feel angry.

Zelin, Adler, and Myerson (1972) designed the ASR to assess both "awareness of anger" and different modes of anger expression. The scores of psychiatric patients on this scale were found to correlate significantly with psychiatrists' ratings of anger. Since the ASR and the RI have each been used in only one or two published studies over the past 25 years, the construct validity of these scales has yet to be established. Although the AI has been used more often in research than other anger measures, Biaggio et al. (1981) found no significant correlations of this scale with either self or observer ratings of anger and hostility and reported that the test-retest stability of the AI over a brief 2-week interval was only .17. In a series of studies, Biaggio and her colleagues (Biaggio, 1980; Biaggio & Maiuro, 1985; Biaggio et al., 1981) examined and compared the reliability, concurrent and predictive validity, and the correlates of the BDHI, RI, AI and ASR. On the basis of their findings, they concluded that evidence of the validity of psychometric measures of anger and hostility was both fragmentary and limited.

A common problem with existing measures of anger and hostility is that, in varying degrees, the experience and expression of anger is confounded with situational determinants of angry reactions. Furthermore, none of these measures explicitly takes the state-trait distinction into account. The ASR Awareness subscale comes closest to examining the extent to which subjects experience angry feelings, but this instrument does not assess the intensity of these feelings at a particular time. A number of BDHI items specifically inquire about the *frequency* that anger is experienced or expressed (e.g., "I *sometimes* show my anger"; "I *never* get mad enough to throw things," *italics added*). While these items implicitly assess individual differences in anger as a personality trait, most BDHI items seem to evaluate hostile attitudes (e.g., resentment, negativism, suspicion), rather than angry feelings. A coherent theoretical framework that distinguishes between anger, hostility, and aggression as psychological concepts, and that takes the state-trait distinction into account, is essential for constructing and validating psychometric measures of anger and hostility.

23.1.2 The State-Trait Anger Scale (STAS)

Anger, as a psychological construct, refers to phenomena that are both more fundamental and less complex than hostility and aggression. The *State-Trait Anger Scale* (STAS: Spielberger, 1980), which is analogous in conception and similar in format to the *State-Trait Anxiety Inventory* (STAI: Spielberger, 1983), was constructed to measure the intensity of anger as an emotional state and individual differences in anger proneness as a personality trait. Prior to constructing the STAS, working definitions of state and trait anger were formulated. State anger (S-Anger) was defined as a psychobiological state or condition consisting of subjective feelings that vary in *intensity*, from mild irritation or annoyance to intense fury and rage, with concomitant activation or arousal of the autonomic nervous system. Trait anger (T-Anger) was defined in terms of individual differences in the *frequency* that S-Anger was experienced over time, assuming that persons high in T-Anger perceive

a wider range of situations as anger-provoking (e.g., annoying, irritating, frustrating) than those low in T-Anger, and more frequently experience elevations in S-Anger whenever such conditions are encountered.

With these working definitions of S-Anger and T-Anger, a pool of items was assembled to assess the intensity of angry feelings (S-Anger) and individual differences in anger proneness (T-Anger). The following are examples of S-Anger items: "I feel angry," "I am furious," "I feel irritated." Examinees report the *intensity* of their angry feelings at a particular time by rating themselves on the following 4-point scale: "Not at all," "Somewhat," "Moderately so," "Very much so." examples of T-Anger items are "I have a fiery temper," "I fly off the handle," "It makes me furious when I am criticized in front of others." In responding to the T-Anger items, examinees indicate how they *generally* feel by rating themselves on the following *frequency* scale: "Almost never," "Sometimes," "Often," "Almost always."

The preliminary form of the STAS, consisting of 15 S-Anger and 15 T-Anger items, was administered to a large sample of university students. Alpha coefficients for the preliminary STAS S-Anger and T-Anger scales were .93 and .87, respectively, providing strong evidence of the internal consistency of both measures. Item-remainder correlations for the individual S-Anger and T-Anger items were also uniformly high (median $r = 0.68$). Jacobs, Latham, and Brown (1988) examined the stability of the STAS for a large group of university students. Test-retest reliability coefficients for the STAS T-Anger scale over a 2-week interval were .70 and .77 for males and females, respectively. In contrast, the stability coefficients for the STAS S-Anger subscale were much lower (.27 for males and .21 for females), as would be expected for a transitory state anger measure.

Given the high internal consistency of the preliminary STAS scales, it was possible to reduce the length of these scales to 10 state and trait items without unduly weakening their psychometric properties. In revising the STAS, it was also considered desirable to develop internally consistent measures of S-Anger and T-Anger that were relatively independent of anxiety. Therefore, in selecting the final set of 10 S-Anger and 10 T-Anger items, those items with the highest item-remainder correlations for each scale and the lowest correlations with measures of anxiety were identified (Barker, 1979).

The STAS S-Anger and T-Anger items were generated primarily on a rational basis. The high internal consistency of these scales, as reflected in item-remainder correlations and alpha coefficients, provides impressive evidence of the utility of the working definitions that guided the item-selection process. Correlations between the 10- and 15-item S-Anger and T-Anger scales ranged from 0.95 to 0.99 for Navy recruits and college students, indicating that the 10-item scales provide essentially the same information as the longer forms (Spielberger, 1988). After eliminating the items with the highest correlations with anxiety, the correlations of the 10-item S-Anger and T-Anger scales with the STAI anxiety scales were substantially lower.

Factor analyses of the STAS S-Anger items identified only a single underlying factor for both males and females, indicating that the S-Anger scale measures a unitary emotional state that varies in intensity. In contrast, factor analyses of the T-Anger items identified two correlated factors, which were labeled Angry Temperament (T-Anger/T) and Angry Reaction (T-Anger/R). The T-Anger/T items describe individual differences in the disposition to express anger, without specifying any provoking circumstance (e.g., "I am a hotheaded person"). The T-Anger/R items describe angry reactions in situations that involve frustration and/or negative evaluations (e.g., "It makes me furious when I am criticized in front of others"). The results of a study of hypertensive patients clearly demonstrated that the two T-Anger subscales measure different facets of trait anger (Crane, 1981). The hypertensive patients had significantly higher T-Anger/R scores than medical and surgical patients with normal blood pressure, but no difference was found in the T-Anger/T scores of these patients.

23.1.3 Concurrent and Construct Validity of the STAS

The concurrent validity of the STAS T-Anger scale was evaluated by computing correlations with the Buss–Durkee (1957) Hostility Inventory (BDHI) and the Hostility (Ho; Cook & Medley, 1954) and Overt Hostility (Hv; Schultz, 1954) scales of the Minnesota Multiphasic Personality Inventory. Moderately high correlations of the STAS T-Anger scale with the three hostility measures were found for large samples of college students and Navy recruits, providing evidence of a strong relationship between T-Anger and hostility.

In a series of studies at Colorado State University, Deffenbacher (1992) and his colleagues used the STAS T-Anger Scale to assess multiple aspects of anger. Individuals with high T-Anger scores reported that they experienced greater intensity and frequency of anger and related physiological symptoms than persons low in T-Anger across a wide range of provocative situations. When provoked, persons with high T-Anger scores also showed stronger tendencies to both express and suppress anger and more dysfunctional coping, as manifested in physical and verbal antagonism. In a study of trait anger and self-concept, Stark and Deffenbacher (1986) found that high T-Anger students did not like themselves as much as those low in T-Anger and did not feel as worthwhile or confident. Negative events such as failure also seemed to have a more devastating impact on high T-Anger individuals (Story & Deffenbacher, 1985), who reported that they experienced high levels of anxiety more frequently than students with low T-Anger scores.

As our research on anger has progressed, the critical importance of differentiating between the experience, expression, and control of anger has become increasingly apparent (Spielberger et al., 1985). It seemed essential not only to distinguish, both conceptually and empirically, between the intensity of the experience of anger as an emotional state (S-Anger) and individual differences in anger proneness as a personality trait (T-Anger), but also to identify and measure the characteristic ways in which people express and control their anger.

23.2 Research on the Experience, Expression, and Control of Anger

The importance of anger has been recognized since ancient times as a fundamental negative emotion provoked by the perception of being attacked or treated badly. Recent research on Type A Behavior and heart disease (Spielberger et al., 1983; Spielberger et al., 1985; Spielberger & London, 1982), demonstrating that anger was the lethal component of the Type A Syndrome, and findings that anger was associated with elevated blood pressure and hypertension have stimulated interest in the development of measures of anger and hostility (Spielberger, 1976, 1980, 1988, 1999).

Observations of daily life and recent research findings suggest that problems with anger are ubiquitous. In a series of studies, Deffenbacher (1992) and his associates (Deffenbacher, Demm, & Brandon, 1986) found that persons high in anger as a personality trait frequently experienced angry feelings across a wide range of situations (cf. Chapter 18 by Schultz et al., this book). Averill (1982) conducted a large survey study, questioning what the “man on the street” had to say about the antecedents, experience, and reactions to anger-provoking situations. The results of this study indicated that, anger is primarily an interpersonal emotion associated with the attribution of blame. Lazarus (1991) has observed that there must be high goal relevance, obstruction of a goal, and a threat to ego identity for anger to be experienced.

A review of the literature identified anger, hostility, and aggression as overlapping constructs that we refer to collectively as the AHA! Syndrome. A careful analysis of these constructs indicated that anger was the fundamental component of this syndrome, and that anger was strongly associated

with hostility and often motivated aggressive behavior. In our ongoing efforts to understand anger, it became clear that we needed not only to access anger as an emotional state and as a personality trait but also to measure the expression and control of anger. Further research lead to the development of the State–Trait Anger Expression Inventory (STAXI-2), which includes scales that assess state and trait anger, anger expression, and anger control. The STAXI-2 scales and subscales are described in Table 23.3.

Table 23.3 State–trait anger expression scale (STAXI-2)

State Anger (S-Anger): A psychobiological emotional state consisting of subjective feelings that vary in intensity from mild annoyance or irritation to intense fury and rage.

- a. *Feeling Angry (S-Ang/F):* Anger ranging from feeling annoyed to furious.
- b. *Feel like Expressing Anger Verbally (S-Ang/V):* (yelling or shouting).
- c. *Feel like Expressing Anger Physically (S-Ang/P):* (hitting someone or breaking things).

Trait Anger (T-Anger): Individual differences in the tendency to perceive a wide range of situations as annoying or frustrating and the disposition to respond to such situations with elevations in S-Anger.

- a. *Angry Temperament (T-Anger/T):* Tendency to experience and express anger indiscriminately (“I am a hot-headed person”).
- b. *Angry Reaction (T-Anger/T):* Disposition to express anger when criticized or treated unfairly by others (“When I do a good job and get a poor evaluation, I feel furious”).

Anger Expression and Control Scales

- *Anger-In (Ax/In):* Frequency that angry feelings are held in or suppressed (“When angry or furious, I boil inside but don’t show it”).
 - *Anger-In (Ax/In):* Frequency that angry feelings are held in or suppressed (“When angry or furious, I boil inside but don’t show it”).
 - *Anger-Out (Ax/Out):* Frequency that S-Anger is expressed in aggressive behavior directed toward other people or objects in the environment (“When angry or furious, I slam doors . . . argue with others . . . say nasty things”).
 - *Anger Control-Out (Ax/Con-Out):* Individual differences in the frequency that individuals attempt to control the outward expression of angry feelings (When angry or furious: “I control my temper”; “I keep my cool”).
 - *Anger Control-In (Ax/Con-In):* Individual differences in the frequency that individuals attempt to reduce the intensity of suppressed angry feelings (When angry or furious: “I try to simmer down . . . try to relax . . . try to soothe my angry feelings”).
-

Suppressed anger, as measured by the STAXI Anger-In Scale, has been consistently identified as an important factor in elevated blood pressure and hypertension (e.g., Johnson, Spielberger, Worden, & Jacobs, 1987; Spielberger, Krasner & Solomon, 1988; Spielberger et al., 1985; van der Ploeg, van Buuren, & van Brummelen, 1988). The STAXI has also been used to examine relationships between hardiness, well-being, and coping with stress (Schlosser & Sheeley, 1985), the anger experienced by patients undergoing treatment for Hodgkins disease and lung cancer (McMillan, 1984), the role of anger in Type A behavior (Booth-Kewley & Friedman, 1987; Spielberger et al., 1988), the effects of marijuana use on the experience and expression of anger (Pape, 1986; Stoner, 1988), and in a series of studies of psychological factors that contribute to chronic pain (Curtis, Kinder, Kalichman, & Spana, 1988). Research with the STAXI provides encouraging evidence of the utility of this inventory for assessing anger in diagnoses, treatment planning, and in the evaluation of treatment outcomes (Spielberger et al., 1995; Spielberger et al., 1998). James Moses (1992, p. 52)

described the STAXI as a “specific, sensitive, psychometric instrument” that has “. . . great potential . . . to significantly further our understanding of important stress-based and stress-influenced syndromes.”

References

- Alexander, F. G., & French, T. M. (Eds.) (1948). *Studies in psychosomatic medicine: An approach to the cause and treatment of vegetative disturbances*. New York: Ronald.
- Averill, J. R. (1982). *Anger and aggression: An essay on emotion*. New York: Springer-Verlag.
- Barker, L. R. (1979). *Personality variables as determinants of performance problems of recruits in the U.S. armed forces*. Unpublished master's thesis, University of South Florida, Tampa.
- Bendig, A. W. (1962). Factor analytic scales of covert and overt hostility. *Journal of Consulting Psychology*, 26, 200.
- Biaggio, M. K. (1980). Assessment of anger arousal. *Journal of Personality Assessment*, 44, 289–298.
- Biaggio, M. K., & Maiuro, R. D. (1985). Recent advances in anger assessment. In C. D. Spielberger & J. N. Butcher (Eds.), *Advances in personality assessment* (Vol. 5). Hillsdale, NJ: Lawrence Erlbaum Associates, Inc.
- Biaggio, M. K., Supplee, K., & Curtis, N. (1981). Reliability and validity of four anger scales. *Journal of Personality Assessment*, 45, 639–648.
- Booth-Kewley, S., & Friedman, H. S. (1987). Psychological predictors of heart disease: A quantitative review. *Psychological Bulletin*, 101, 343–362.
- Buss, A. H. (1961). *The psychology of aggression*. New York: John Wiley & Sons.
- Buss, A. H., & Durkee, A. (1957). An inventory for assessing different kinds of hostility. *Journal of Consulting Psychology*, 21, 343–349.
- Cook, W. W., & Medley, D. M. (1954). Proposed hostility and pharisaic-virtue scales for the MMPI. *The Journal of Applied Psychology*, 38, 414–418.
- Crane, R. S. (1981). The role of anger, hostility, and aggression in essential hypertension. (Doctoral dissertation, University of South Florida, Tampa, FL, 1981) *Dissertation Abstracts International*, 42, 2982B.
- Curtis, G., Kinder, B., Kalichman, S., & Spana, R. (1988). Affective differences among subgroups of chronic pain patients. *Anxiety Research: An International Journal*, 1, 65–73.
- Darwin, C. (1965). *The expression of emotions in man and animals*. Chicago: University of Chicago Press. (Original work published 1872)
- Deffenbacher, J. L. (1992). Trait anger: Theory, findings, and implications. In C. D. Spielberger & J. N. Butcher (Eds.), *Advances in personality assessment* (Vol. 9). Hillsdale, NJ: Lawrence Erlbaum Associates, Inc. (1992)
- Deffenbacher, J. L., Demm, P. M., & Brandon, A. D. (1986). High general anger: Correlates and treatment. *Behavior Research and Therapy*, 24, 480–489.
- Dembroski, T. M., MacDougall, J. M., Williams, R. B., Haney, T. L., & Blumenthal, J. A. (1985). Components of type A, hostility, and anger-in: Relationship to angiographic findings. *Psychosomatic medicine*, 47, 219–233.
- DiGiuseppe, R., & Tafrate, R. C. (2007). *Understanding anger disorders*. New York: Oxford University Press.
- Diamond, E. L. (1982). The role of anger and hostility in essential hypertension and coronary heart disease. *Psychological Bulletin*, 92, 410–433.
- Dunbar, H. F. (1943). *Psychosomatic diagnosis*. New York: P.B. Hoeber.
- Evans, D. R., & Stangeland, M. (1971). Development of the reaction inventory to measure anger. *Psychological Reports*, 29, 412–414.
- Freud, S. (1959). Why war? In J. Strachey (Ed.), *Collected papers* (Vol. 5). London: Hogarth Press. (Original work published 1933)
- Friedman, M., & Rosenman, R. H. (1974). *Type A behavior and your heart*. Greenwich, CO: Fawcett Publications, Inc.
- Gentry, W. D., Chesney, A. P., Gary, H. G., Hall, R. P., & Harburg, E. (1982). Habitual anger-coping styles: I. Effect on mean blood pressure and risk for essential hypertension. *Psychosomatic medicine*, 44, 195–202.
- Harburg, E., Erfurt, J. C., Hauenstein, L. S., Chape, C., Schull, W. J., & Schork, M. A. (1973). Socio-ecological stress, suppressed hostility, skin color, and black-white male blood pressure: Detroit. *Psychosomatic medicine*, 35, 276–296.
- Jacobs, G. A., Latham, L. E., & Brown, M. S. (1988). Test-retest reliability of the state-trait personality inventory and the anger expression scale. *Anxiety Research*, 1, 263–265.
- Johnson, E., Spielberger, C., Worden, T., & Jacobs, G. (1987). Emotional and familial determinants of elevated blood pressure in black and white adolescent males. *Journal of Psychosomatic Research*, 31, 287–300.
- Lazarus, R. S. (1991). *Emotion and adaptation*. London: Oxford University Press.

- Matthews, K. A., Glass, D. C., Rosenman, R. H., & Bortner, R. W. (1977). Competitive drive, pattern A, and coronary heart disease: A further analysis of some data from the Western Collaborative Group Study. *Journal of Chronic Diseases*, 30, 489–498.
- McMillan, S. C. (1984). *A comparison of levels of anxiety and anger experienced by 2 groups of cancer patients during therapy for Hodgkin's disease and small cell lung cancer*. Unpublished master's thesis, University of South Florida, Tampa.
- Moses, J. A. (1992). State-trait anger expression inventory, research ed. In D. J. Keyser & R. C. Sweetland (Eds.), *Test critiques* (Vol. IX, pp. 510–525). Austin, TX: PRO-ED, Inc.
- Novaco, R. W. (1975). *Anger control: The development and evaluation of an experimental treatment*. Lexington, MA: Lexington Books/D.C. Heath.
- Pape, N. E. (1986). Emotional reactions and anger coping strategies of anger suppressors and expression (Doctoral dissertation, University of South Florida, Tampa, 1986). *Dissertation Abstracts International*, 47, 2627B.
- Russell, S. F. (1981). *The factor structure of the Buss-Durkee hostility inventory*. Unpublished master's thesis, University of South Florida, Tampa.
- Schlosser, M. B., & Sheeley, L. A. (1985, August). *Subjective well-being and the stress process*. Paper presented at the 93rd annual convention of the American Psychological Association, Los Angeles, CA.
- Schultz, S. D. (1954). A differentiation of several forms of hostility by scales empirically constructed from significant items on the MMPI. *Dissertation Abstracts*, 17, 717–720.
- Spielberger, C. D. (1976). Stress and anxiety and cardiovascular disease. *Journal of the South Carolina Medical Association*, (Suppl. 15), 15–22.
- Spielberger, C. D. (1980). *Preliminary manual for the state-trait anger scale (STAS)*. Tampa, FL: University of South Florida, Human Resources Institute.
- Spielberger, C. D. (1983). *Manual for the state-trait anxiety inventory: STAI (Form Y)*. Palo Alto: Consulting Psychologists Press.
- Spielberger, C. D. (1988). *Manual for the state-trait anger expression inventory (STAXI)*. Odessa, FL: Psychological Assessment Resources, Inc.
- Spielberger, C. D. (1999). *Professional manual for the state-trait anger expression inventory-2 (STAXI-2)*. Odessa, FL: Psychological Assessment Resources, Inc.
- Spielberger, C. D., Jacobs, G., Russell, S., & Crane, R. (1983). Assessment of anger: The state-trait anger scale. In J. N. Butcher & C. D. Spielberger (Eds.), *Advances in personality assessment* (Vol. 2, pp. 159–187). Hillsdale, NJ: Lawrence Erlbaum Associates, Inc.
- Spielberger, C. D., Johnson, E. H., Russell, S. F., Crane, R. J., Jacobs, G. A., & Worden, T. J. (1985). The experience and expression of anger: Construction and validation of an anger expression scale. In M. A. Chesney & R. H. Rosenman (Eds.), *Anger and hostility in cardiovascular and behavioral disorders* (pp. 5–30). New York: Hemisphere/McGraw-Hill.
- Spielberger, C. D., Krasner, S. S., & Solomon, E. P. (1988). The experience, expression and control of anger. In M. P. Janisse (Ed.), *Health psychology: Individual differences and stress* (pp. 89–108). New York: Springer Verlag/Publishers.
- Spielberger, C. D., & London, P. (1982). Rage boomerangs: Lethal type-A anger. *American Health*, 1, 52–56.
- Spielberger, C. D., Reheiser, E. C., & Sydeman, S. J. (1995). Measuring the experience, expression, and control of anger. In H. Kassirer (Ed.), *Anger disorders: Definitions, diagnosis, and treatment* (pp. 49–76). Washington, DC: Taylor & Francis.
- Spielberger, C. D., Ritterband, L. M., Sydeman, S. J., Reheiser, E. C., & Unger, K. K. (1998). Assessment of emotional states and personality traits: Measuring psychological vital signs. In J. N. Butcher (Ed.), *Clinical personality assessment: Practical approaches* (pp. 42–58). New York: Oxford University Press.
- Stark, R. S., & Deffenbacher, J. L. (1986, April). *General anger and self-concept*. Paper presented at Rocky Mountain Psychological Association, Denver, CO.
- Stoner, S. B. (1988). Undergraduate marijuana use and anger. *Journal of Psychology*, 122, 343–347.
- Story, D., & Deffenbacher, J. L. (1985, April). *General anger and personality*. Paper presented at Rocky Mountain Psychological Association, Tucson, AZ.
- Tavris, C. (1989). *Anger the misunderstood emotion*. New York: Touchstone.
- van der Ploeg, H. M., van Buuren, E. T., & van Brummelen, P. (1988). The role of anger in hypertension. *Psychotherapy and Psychosomatics*, 43, 186–193.
- Williams, R. B., Jr., Barefoot, J. C., & Shekelle, R. B. (1985). The health consequences of hostility. In M. A. Chesney & R. H. Rosenman (Eds.), *Anger and hostility in cardiovascular and behavioral disorders* (pp. 173–185). Washington, DC: Hemisphere.
- Zelin, M. L., Adler, G., & Myerson, P. G. (1972). Anger self-report: An objective questionnaire for the measurement of aggression. *Journal of Consulting and Clinical Psychology*, 39, 340.

Chapter 24

“Righteous” Anger and Revenge in the Workplace: The Fantasies, the Feuds, the Forgiveness

Thomas M. Tripp and Robert J. Bies

Abstract Revenge is part of the social fabric of organizational life. For many, revenge is typically viewed as an irrational, if not evil, response, to events in the workplace. However, there is an emerging scholarly view of revenge that departs from that conventional wisdom. This view is what we refer to as “revenge as justice.” In this chapter, we review a growing body of research across academic disciplines that finds the motivation for revenge is, more often than not, grounded in a perception that one has been the victim of *undeserved* harm and feelings of injustice. Drawing on empirical findings, we argue that revenge is not motivated by mere anger grounded in frustration, but a *righteous* anger, an emotional response to correct and prevent injustice. As such, revenge is central to the process of justice in organizations. While righteous anger is core variable in our analysis, we illustrate how cognitive mistakes and biases can shape the emotion of righteous anger and the act of revenge. Finally, we argue that there is a rationality and morality to revenge, which must be understood through emotional lens of righteous anger.

I was so angry. My boss played favorites and promoted a friend of hers to a job everyone knows that I deserved to get. I had dreams of making her feel pain, deep pain... the same pain I was feeling. (Middle manager, Telecommunications Company)

When he betrayed me by sharing confidential information to the CEO, I knew I had to get even. I bad-mouthed him, and I sabotaged his work. And I am not done yet. He deserves the justice that I will be serving. (Senior-level manager, Financial Services Company)

He publicly berated me, humiliated me in front of the team. I never felt so angry. I hated him, I wanted him to die. I lived with that hatred for months until it literally ate my insides out. At that moment, I realized that he had been victimizing me for all of those months as I obsessed with what he did to me. With my friends and co-workers help, I let go. I chose to forgive him. I have not forgotten what he did, but the anger does not eat me up anymore. (Middle manager, Energy Company)

Revenge is a phenomenon that both fascinates us and leaves us fearful. At some moment in our lives, we have all felt the primal urge to “get even” when harmed, and the passion of that moment makes revenge feel like the “right” thing to do. It is not only when we are harmed personally that revenge feels like the righteous response; for, we vicariously experience that righteous feeling when others are harmed or when swift vengeance is dealt to the perpetrator of harm (Jacoby, 1983; McLean-Parks, 1997). Indeed, witness the audience’s applause and approval of acts of revenge in such films as *Dirty Harry* and *The First Wives Club*.

T.M. Tripp (✉)
Washington State University at Vancouver, Vancouver, WA, USA
e-mail: ttripp@vancouver.wsu.edu

But we are also afraid of revenge. We are fearful of the sometimes uncontrollable power of the passion of revenge (Tripp & Bies, 1997), when such emotions can cause events to escalate out of control, as in a feud or physical violence (Bies & Tripp, 1996). Our fears about revenge are further magnified when we witness how revenge has shaped the centuries of hatred and hostility in nations around the world, as in the case of the Balkans (Pomfret, 1995).

Recently, amidst the growing scholarly research on revenge in the workplace, there is an emerging view of “revenge as justice” (Bies & Tripp, 1996; McLean-Parks, 1997; Tripp & Bies, 1997). While it is true that revenge is typically viewed as an irrational, if not evil, response, it is also true that the motivation for revenge is often rooted in the perception of *undeserved* harm and feelings of injustice (Tripp & Bies, 1997; McLean-Parks, 1997). Given the concept of “deservingness” is central to both normative formulations of justice (Feinberg, 1974) and social psychological theories of justice (e.g., Adams, 1965; Crosby, 1976), attempts to “get even” or “evening the score” – that is, to receive what one rightfully deserves – are also central to justice. Moreover, there is a tradition in philosophy (e.g., Solomon, 1990) and legal theory (e.g., Cahn, 1949) that recognizes that the emotions of injustice are central to understanding the justice process. Yet, the dominant models of organizational justice do not view revenge as central to the justice process.

In this chapter, we argue that *revenge is central to the process of justice* in organizations, because it reflects a response intended to remedy or prevent injustice. In focusing on revenge as justice, emotions become figural in understanding responses to injustice (Bies, 1987, 2006). While we highlight the importance of the emotions of injustice, we argue that there is a rationality and morality to revenge. Needless to say, this perspective that revenge is central to a theory of justice is a radical departure from current models.

24.1 Righteous Anger and Revenge: It Begins with the Sense of Injustice

Our analysis begins with the premise that, to understand righteous anger and revenge in the workplace, one must understand the events that arouse the sense of injustice – which are the emotions motivating revenge (Bies, 1987, 2001). Choosing the sense of injustice as the starting point for analyzing justice dynamics has its intellectual roots in the seminal legal theory of Edmond Cahn. In his book, *The Sense of Injustice* (1949), Cahn asks: “Why do we speak of the ‘sense of injustice’ rather than the ‘sense of justice’?” Cahn answers: “Because ‘justice’ has been so beclouded by natural-law writings that it almost inevitably brings to mind some ideal relation or static condition or set of perceptual standards, while we are concerned, on the contrary, with what is active, vital, and experiential in the reactions of human beings” (p. 13).

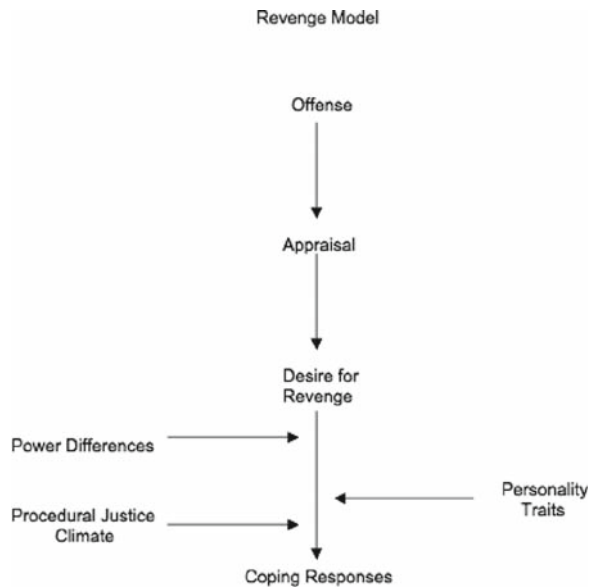
For Cahn, justice is “not a state, but a process; not a condition, but an action. ‘Justice,’ as we shall use the term, means the *active process* of remedying or preventing that which would arouse the sense of injustice” (p. 13). He defines the sense of injustice as “the sympathetic reaction of outrage, horror, shock, resentment, and anger, those affections of the viscera and abnormal secretions of the adrenals that prepare the human animal to resist attack. Nature has thus equipped all men to regard injustice to another as personal aggression” (p. 24).

Building on Cahn’s argument, the sense of injustice is aroused by the provocation of another person. The sense of injustice is a response to a *perceived harm or wrongdoing* by another party (Tripp & Bies, 1997). It is this provocation that elicits the anger, which motivates the revenge (Bies & Tripp, 1996; McLean-Parks, 1997). Indeed, across a series of studies, those who engage in revenge always report taking action in response to the *provoking* action of another person (see Tripp & Bies, 1997, for a review of this evidence). As such, revenge is not a random response, but an intentional

and directed response to perceived harm or wrongdoing. To elaborate, not only is revenge rooted in an intuitive “sense of injustice,” but it usually reflects a self-controlled response. Indeed, in many cases, revenge is a “cool and calculated” response (Tripp & Bies, 1997).

In our research program on revenge in the workplace, we have found that righteous anger plays a central role in the link between injustice and revenge. In fact, in our model (see Fig. 24.1) anger mediates the relationship between actions (i.e., provocations, such as injustice) and responses (e.g., revenge, forgiveness, reconciliation, and avoidance). We label these responses as *coping* responses, precisely because it is their circumstances and anger that employees are trying to cope with when they choose to, for example, get even or forgive.

Fig. 24.1 Revenge model



Before exploring the details of righteous anger and the appraisal process in depth, we summarize our model of revenge. The model describes the episodic features of anger and revenge, in somewhat of a chronological order, while also describing the workplace context in which anger plays out. After outlining our model, we will examine social cognitive dynamics that shape and intensify righteous anger and revenge as well as fueling hatred and feuding in the workplace.

24.2 Our Model: From Provocation to Righteous Anger to Revenge

Our model of revenge begins with the perception of some offense, which triggers a blame-placing, appraisal process. If blame is assigned to someone, that attribution motivates righteous anger and the desire for revenge. We then identify how people act out the desire for revenge, which we refer to as coping responses. We also identify moderators of those coping responses. Note that many of these same issues are dealt with in psychological models of emotion which focus on anger rather than revenge and which use the term appraisal in a broader sense (e.g., Chapter 15 by Wranik and Scherer, this book).

24.2.1 Offense

First, the episode begins with some sort of offense. That is, another employee or manager offends the “victim.” We have found (Bies & Tripp, 2004; Aquino, Tripp, & Bies, 2006) that offenses fall into three categories: (a) goal obstruction; (b) violation of rules, norms, and promises; and (c) status and power derogation.

24.2.1.1 Goal Obstruction

When one prevents the achievement of another’s goals in the workplace, the prevention can lead to acts of revenge in response (Morrill, 1992). Goal obstruction can lead to frustration (Buss, 1962), and that frustration can lead to an aggressive response like revenge (Neuman & Baron, 1997).

24.2.1.2 Violation of Rules, Norms, or Promises

Employees become angered when the formal rules of the organization are violated (Bies & Tripp, 1996). One such example is employees who act as if the rules do not apply to them, thus obtaining special benefits or avoiding common burdens. An example of norm violation is a teammate who shirks his duties or otherwise does not carry his weight while receiving all the rewards that accrue to the team. More broadly, any perceived inequities on the job or violations of fairness norms can motivate revenge (cf. Skarlicki & Folger, 1997).

24.2.1.3 Status and Power Derogation

Actions that diminish another’s reputation or power can motivate revenge (Bies & Tripp, 1996). For example, destructive criticism (Baron, 1988), public ridicule intended to embarrass a subordinate or coworker (Morrill, 1992), or when the employee is accused wrongly by boss or peer (Bies & Tripp, 1996) can motivate victims to regain status and power through attacking those who harmed them.

Note that victims view the latter two categories as instances of injustice (Aquino et al., 2006). That is, they perceive such acts as unfair, because these acts result in breaches of procedure or in mistreatment of employees. The organizational justice literature refers to such injustices as “procedural injustice” and “interactional injustice,” respectively. The justice literature has spent decades uncovering just what workplace acts employees perceive as unfair, but relatively much less time understanding what employees *do* about injustices *after* they perceive them. One thing employees do is seek justice through revenge (Jacoby, 1983; Tripp, Bies & Aquino, 2007).

24.2.2 Appraisal Process

Whether an anger-arousing offense that *can* motivate revenge actually *does* motivate revenge depends on a variety of social–cognitive factors (Bies, Tripp, & Kramer, 1997). In our model, an offense triggers a search for causal explanation as to “why” the event occurred (Wong & Weiner, 1981). In this causal analysis, people will search for many factors – some factors that may “discount” the harmdoer’s responsibility for the action and some factors that may exaggerate the harmdoer’s responsibility for the action (Kelley, 1972). For example, there may exist mitigating circumstances that create a “reasonable doubt” in people’s minds. In this case, we predict that revenge is less likely (Bies & Tripp, 1996). Alternatively, there may exist compounding and amplifying circumstances, such as social support that reinforces early, paranoid beliefs about the harmdoer’s motives as overly

sinister (Bies & Tripp, 1996). In this case, we would predict that revenge is more likely. In general, whether the offense arouses the sense of injustice and associated emotions of outrage or anger will depend on the blame that the causal analysis produces (Bies, 1987; Cohen, 1982; Utne & Kidd, 1980).

Of particular importance in the causal analysis is the perception of intentionality (Thomas & Pondy, 1977; Neuman & Baron, 1997). If intentionality is determined, then that causal judgment will magnify the perception of unfair treatment (cf. Garrett & Libby, 1973; Leventhal, Weiss, & Long, 1969). Moreover, if the causal analysis leads to a judgment that the harm was intentional, the desire for revenge will be triggered, and revenge behavior will be more likely (Bies et al., 1997). On the other hand, if the action is viewed as unintentional, it can influence the harmed party to act in a conciliatory manner toward the harmdoer (Baron, 1990).

Following this line of reasoning, how the sense of injustice is transformed into revenge depends on how one makes sense of, or cognitively processes, the harm or wrongdoing. As part of this *sense-making process* (Weick, 1995), the assignment of blame is critical. If one can place blame on another person, pain and confusion converts to anger and determination, and thus revenge becomes more likely. It is the assignment of blame that is at the foundation of the rationality and morality of revenge.

In taking a sense-making perspective (Weick, 1995), we begin with the premise that there is no necessary relationship between the “objective” characteristics of an incident or situation and the emergence of revenge (Bies et al., 1997). Whether the situation evolves into revenge and social conflict depends on parties’ conceptualization of the situation (Deutsch, 1975; Thomas, 1976). Of particular importance in the conceptualization process is the parties’ assessment of each other’s motives and intentions (Thomas & Pondy, 1977; Neuman & Baron, 1997). For example, assuming an action is detrimental to one party, the other party is more likely to get angry when the action is perceived as intentional rather than unintentional, and perpetrated for socially unacceptable rather than socially acceptable reasons (Averill, 1982). Moreover, such feelings of anger are more likely to trigger an aggressive response, like revenge, on the part of the affected party (Ferguson & Rule, 1983), which may induce or escalate conflict. In other words, how the parties involved perceive a dispute in *psychological* terms is a critical factor explaining conflict.

A similar line of reasoning is found in the seminal work of Felstiner, Abel, and Sarat (1980–1981), who proposed: “disputes are not things; they are social constructs. . . [that] exist only in the minds of the disputants” (pp. 632–633). Whether an incident is “transformed” into revenge would depend on the parties’ interpretation of the event. This interpretation process has three sequences, which Felstiner et al. identify as “naming, blaming, and claiming.” In a similar perspective, Sheppard, Lewicki, and Minton (1992) argue that how one “perceives, defines, and interprets a dispute is often more critical than the substantive nature of the dispute itself” (p. 47). The cognitive and social factors that shape this interpretation process are discussed below.

Bies and Tripp (1996) and Bies et al. (1997) identified two kinds of motives in the more intentional attributions – *selfishness* and *malevolence*. A selfish harmdoer causes harm for personal profit, picking the victim purely based on opportunity. Sometimes, the victim is picked as a “mark” – i.e., somebody who has something the harmdoer wants or is somehow integral to the harmdoer’s goals. Here the harm is an act of commission in that the victim is consciously targeted. Other times, the victim is not targeted at all: the victim is not in the harmdoer’s “equation” and thus the victim’s being harmed is just an unfortunate side effect of the harmdoer’s goal-directed behavior. Here, the harm is act of *omission*; the victim was in the way, and the harmdoer chose not to stop or otherwise avoid the victim.

A *malevolent* harmdoer causes harm for the sake of inflicting pain on the victim. Here, the act is always commissive because the victim is always consciously targeted. Malevolent harm differs from

selfish harm: the malevolent harmdoer targets the victim not because the victim has something the harmdoer wants, but because the harmdoer enjoys making *that particular* victim feel pain. That is, here it truly is “personal.” Malevolent harm is, by our definition, the most intentional attribution one can make about the motives of the harmdoer.

Crossley (2006), in both scenario-based experimental and critical-incident field studies, showed that victims do distinguish between selfishness and malevolence. Moreover, he found that which motive victims perceived affects their responses: malevolence more than greed angers victims, and anger increases revenge and avoidance while decreasing reconciliation.

Unfortunately, much evidence exists that such sense-making processes about conflict and blame are biased – i.e., they produce more blame than the harmdoer usually deserves. The exaggerated blame comes from an obsessive, self-centered, and ego-defensive process. Victims spend an inordinate amount of time piecing together what happened and what should be done. The victims see the conflict from primarily their own perspective, where they are the central players. Moreover, victims choose beliefs that bolster their self-esteem such that conflict is usually the *other* party’s fault, not the victim’s fault. The other party is the hateful aggressor, whereas oneself is the sympathetic victim.

To better understand the sense-making process, it is helpful to identify the individual biases. These biases include *hypervigilance* and *rumination* (Bies et al., 1997), *exaggerated self-reference* (Kramer, 1995), *exaggerated perceptions of conspiracy* (Kramer 1995), *actor–observer bias* (Jones & Nisbett, 1972), *sinister attribution error* (Kramer, 1995), *biased punctuation of conflict history* (Kramer, 1995), *social information* (Asch, 1951; Salancik & Pfeffer, 1978), and *confirmation bias* (Hastorf & Cantril, 1954). These biases, we will argue, act as “attention-focusing” devices: they focus the attention of the victim-cum-avenger on subsets of information regarding the possible motivations of harmdoers. In short, these devices determine to whom and how much the victim-cum-avenger assigns blame.

24.2.2.1 Hypervigilance

Numerous organizational theorists (Janis, 1983; Pfeffer, 1992; Weick, 1995) have noted the adaptive role that social vigilance plays within organizations. Given the often competitive and political nature of organizational life, vigilance increases the likelihood that individuals will detect threats and opportunities, and respond effectively to them (Morrill, 1992). *Hypervigilance*, however, represents an extreme and less adaptive mode of perception in which individuals over-process information, prompting the drawing of erroneous inferences from it (Janis, 1983). For the hypervigilant organizational actor who perceives himself or herself as harmed or threatened, every social interaction becomes scrutinized for hidden meaning and sinister purpose. Thus, the hypervigilant avenger dissects every act, no matter how seemingly benign, for hints of insult, humiliation, and derogation. From this vantage point, even the meaningless averted glance or failure to return a greeting takes on sinister significance and malevolent import, thus increasing the likelihood of revenge.

24.2.2.2 Rumination

Rumination involves the negative framing and editing of social information. Empirical studies have shown that rumination following negative events tends to increase negative thinking about those events and also prompts a pessimistic attribution style when trying to explain why they happened (Pyszczynski & Greenberg, 1987; Lyubomirsky & Nolen-Hoeksema, 1993; Kramer, 1995). Somewhat ironically, rumination also appears to increase individuals’ confidence in their interpretations, further exacerbating the problem of reality testing (Wilson & Kraft, 1993). Thus, the more

aggrieved avengers ruminate about the insults they have experienced, the more convinced they become that the insults were intentional and signify larger and hidden threats.

24.2.2.3 Exaggerated Self-Reference

Exaggerated self-reference is one’s belief that others’ action(s) involve oneself, even though there is insufficient information to confirm, or even suggest, that the others’ action(s) regard oneself at all (Kramer, 1995). By “involve” and “regard” we mean that the others’ action(s) are caused by or intended to impact oneself. For example, suppose one day while walking down the hall past an open office, the managers in the office close the door as the employee walks by. The employee then reflects upon the incident, hypothesizing the possible reasons why the managers shut the door *just then*. When the employee concludes, “they didn’t want me to hear their conversation, probably because they were talking about me,” the employee has ignored or dismissed plausible alternative hypotheses, such as “they didn’t want *anyone* to hear” or “it was a coincidence.” Basically, the employee has favored the self-referential explanation over the non-self-referential explanations, even though the latter may be more likely.

We predict that exaggerated self-reference leads to an overly intentional attribution where malevolent motives are assigned to benign or random events. For example, Kramer (2001) provides the example of how when an employee spends much of one’s time thinking about the boss, the employee assumes, incorrectly, that the boss is spending nearly as much time thinking about the employee. Thus, when the boss’s actions affect the employee, the employee is certain that the effect was intended. However, often the effects are accidental, as the boss, who is busy thinking about her boss, neglected to consider how the employee might be affected.

24.2.2.4 Exaggerated Perceptions of Conspiracy

Another perceptual pattern that contributes to the perceived need to engage in revenge behavior is the *exaggerated perception of conspiracy* associated with paranoid cognitions (Colby, 1981). This term refers to the paranoid perceiver’s tendency to view the actions of others in the organization as more tightly connected or coordinated than they actually are. That is, the paranoid perceiver “connects the dots,” even when randomly placed dots should not be connected. For example, Anita is turned down for promotion to project leader of a cross-functional team, she is told, “in part because some teammates question your ability to lead.” Anita, feeling stunned and hurt, begins entertaining paranoid thoughts about who it was and how many it was, and even whether they coordinated their negative recommendations. She might think, “it wasn’t just Sally in Marketing who recommended I not get the promotion, but I bet Ralph and Diego badmouthed me too, and at least they all probably talked about it.” This may lead the victim to then question Diego, “Did you know what Sally was going to do? Did you encourage her?”

24.2.2.5 Actor–Observer Bias

The actor–observer bias (Jones & Nisbett, 1972) represents another bias in the causal analysis that can shape the motivation for revenge. When attributing blame to a *particular* actor, how individuals attribute motives depends upon whether the individuals are (a) the actors, themselves, judging their own actions, or (b) mere observers, judging someone else’s actions. Specifically, when actors judge their own bad actions, the actors favor external or environmental causes; yet, when observers judge the actions of some other actor, the observers favor internal, trait-based causes. For example, an employee judging her own work performance might say, “I’m not working hard because performance

isn't tied to pay, and the pay is so little anyway." Whereas a coworker observing and judging the employee's behavior might conclude, "She's not working hard because she's a lazy person." Pay is an external attribution while laziness is an internal attribution.

In assigning blame in conflict, the actor–observer bias may play an important role. Victims are biased to judge the motivations behind the harmdoer's harms as being internal, and thus intentional. Therefore, fewer harmdoers will be spared the benefit of the doubt that they may have been forced to harm the victim or that it was just bad luck. Moreover, when judging one's own harms toward the other, one is biased to judge one's own motivations as external, and thus not intentional.

Road-rage provides a good example of the actor–observer bias in conflicts. When one driver is "cut off" in traffic by another driver and has to slam on the brakes to avoid an accident, the first driver may scream at the other driver "you incompetent jerk!" or something like that. Incompetence and jerkiness are *internal, trait* attributions. That is, the first driver, knowing *nothing* about the second driver except that the second driver cut off the first driver – after all, they just met – is *certain* the second driver possesses these traits. The second driver, trying to determine how he just cut off the first driver, wonders aloud, "Did I just do that? I wish I'd gotten more sleep last night so that I can pay more attention like I usually do." The second driver never exclaims, "I am such a jerk!" Lack of sleep is an *external* attribution; the second driver avoids the internal attributions of aggressiveness and incompetence.

24.2.2.6 Sinister Attribution Error

Given such biases and errors, it is no surprise then that people also succumb to the "sinister attribution error." That is, when individuals over-attribute sinister and malevolent motives to others' actions (e.g., "she wasn't just being careless or even just selfish; she was mean-spirited"), they may perceive harmful intent or believe they are being belittled even in their otherwise seemingly benign social encounters (Kramer, 1994; 1995). This error occurs even in the face of ambiguous information about another's motives, when rational people would decide they just cannot know what the harmdoer's motives were. But people are not rational and do not decide they cannot know; rather, they conclude that motives must have been hostile and sinister (Kramer, 1995).

24.2.2.7 Confirmation Bias

Reinforcing this sinister attribution error is the *confirmation bias* (Hastorf & Cantril, 1954). The confirmation bias is the tendency for people to seek only information that proves their initial opinions and hypotheses correct. Yet, disconfirmatory information is more logically useful – i.e., more diagnostic. Rational decision-makers should seek both kinds of information and place more weight on disconfirmatory information (Wason, 1960). Thus, when harmed, victims should seek to disconfirm their initial, suspicious attributions. However, victims, like all people, are biased toward seeking confirmation of their worst, suspicious fears, looking to prove true their paranoid theories. For example, Diana gets fired, she suspects, because a customer complained about her to her boss. Diana then goes looking for evidence that the customer complained, including trying to contact the customer, asking coworkers about the customer, and interrogating her boss about whether the boss met with the customer. What Diana does not look for, however, is evidence that she was fired for another reason, such as chronic insubordination and countless service errors.

24.2.2.8 Social Information

So far, we have argued that victims prematurely develop sinister explanations and then set out to confirm those explanations. They do not do this in social isolation. Rather, these judgments and

associated emotions often get reinforced, and sometimes attenuated, by the victims’ social support networks. Our data reveal that quite often victims discuss their feelings and hypotheses surrounding the harmdoers’ actions in the company of other people, often coworkers. Indeed, Morrill (1992) describes workplace social gatherings where the purpose is to discuss managerial mistakes and wrongdoings as “bitch sessions.” Goldman (2003) studied the etiology of EEOC claims filed by terminated employees against their former employers. He found that a strong predictor of whether claims were filed was the process by which attorneys and family members discussed offenses with the victim.

“Bitch sessions” open up the attribution process to group dynamics, such as conformity and group think. When unsure of the true nature of causality, the victim may adopt the group’s opinion – more so when the group has consensus (Ross & Nisbett, 1992). Furthermore, the “bitch session” process may produce, in the victim’s eyes, an illusion of consensus. That is, social support entails, among other things, empathic support, which friends often provide unconditionally. It is very easy to misconstrue empathic support for agreement with one’s suspicious opinions. Thus, victims may find more reinforcement for their early, suspicious attributions than really exists. Indeed, other people may provide “cues” to lead the victim to a conclusion (Salancik & Pfeffer, 1978), thus “priming” the victim to seek revenge.

Alternatively, a few opposing opinions – that the harmdoer’s motives really are benign – may be enough to convince the victim not to make personalistic attributions. In experiments on social conformity (e.g., Asch, 1951), the presence of only a small percentage of dissenters was enough to change the subjects’ opinions and actions. Social accounts by harmdoers may have a similar impact on muting the desire for revenge (Bies, 1987).

An interesting question is: when the victims are actively seeking information, to whom do they turn? Do they turn to their friends, who know them well but may or may not know the harmdoer well? Or do they turn to the harmdoer’s friends? The harmdoer’s friends may be the best source for information because they most likely have the best access to the harmdoer’s actual thinking. Nonetheless, we predict that the victims turn to their own friends because their friends will provide much more sympathetic emotional support than would the harmdoer’s friends and are more likely to confirm their initial suspicions than are the harmdoer’s friends. Even worse, perhaps, within their group of friends and coworkers, the victims may seek out those who dislike the harmdoer and who are very willing to confirm any opinion that criticizes the harmdoer.

In summary, the appraisal process is quite error-prone. That is, victims make attribution errors about the harmdoer’s motives, some quite predictable. All such errors cumulatively add up to a sloppy appraisal in which people excessively blame the harmdoer, believing that the harmdoer’s intentions were more intentional and personal than they actually were.

24.2.3 Righteous Anger and the Desire for Revenge

Blame leads to righteous anger. In our earlier research where we content-analyzed nearly 600 stories of workplace revenge, as well as in others’ research (Hornstein, 1996; Mikula, 1986; Mikula, Petri, & Tanzer, 1990), the nature of the anger became clear. As we detailed in a 2001 publication (Bies & Tripp, 2001), the emotions of injustice can be characterized in terms of a variety of facets. These facets include *surprise*, *intensity*, *moral righteousness*, *fear*, and *rumination*.

24.2.3.1 Surprise

Most acts of harm that provoke revenge come as a surprise to the victim. They simply did not see harms coming. Thus, victims often report feeling “confused” or “stunned” by the harm. As one

victim reported in Bies and Tripp (1998, p. 210), “I couldn’t believe what had just happened. I trusted him. When he attacked me in front of my co-workers, I was paralyzed and speechless.”

24.2.3.2 Intensity

Victims often focus on the intensity of the emotions they experience in injustice. The intensity reveals a strong visceral response of physiological and psychological pain. In fact, the initial emotions of injustice are often described as “white hot,” “furious,” “bitter,” and volatile, characterized by expressions of pain, anger, and rage (Bies & Tripp, 1996). One person described herself as “inflamed” and “enraged” and “consumed” by thoughts of revenge, while another person needed to satisfy the “burning desire of revenge.” Others have reported a variety of physiological symptoms including uncontrollable crying, “knots in the stomach,” and physical exhaustion. Based on these findings, it is clear that the righteous anger is an intensely *felt* experience. The anger is experienced not just psychologically, but also *physiologically*.

24.2.3.3 Moral Righteousness

Victims are more than stunned and mad, they moralize about their experience. They were not just hurt; they were *wronged* (Bies & Tripp, 1996). That is, they focus on the harm as being a violation of norms of fair treatment. Given the types of injustice that provoke revenge, especially status derogation and rule violations, it is not surprising that victims’ anger often reflects a sense of violation that was more than mere “unmet expectations.” For example, in Bies and Tripp (1996), one individual described a betrayed confidence as causing her world to be “shattered,” as what she assumed to be “sacred and true – the trust of a friend” was violated, if not destroyed forever. It is the sense of injustice that commands their attention. And it is this sense of injustice which they feel must be balanced and quenched with an act of revenge.

It is the righteousness of this anger that gives legitimacy to acts of revenge (Bies, 1987). The claim of injustice legitimates the act of revenge, as there is moral justification for the act of revenge. Indeed, Tripp, Bies, and Aquino (2002) find that people are more likely to view injustice-motivated revenge as legitimate relative to revenge motivated out of self-interest.

24.2.3.4 Fear

Several respondents in Bies and Tripp (1998) reported being “fearful for their well-being,” after being victimized by their bosses. One person reported, “I would stay at my desk and not even go to the bathroom, for the fear, if I was away from my desk, my boss would ‘hammer’ me in public” (p. 210). Not surprisingly, such people reported feeling vulnerable and powerless, for the boss’s tyranny had “broken the spirit and willingness to fight back,” as described by one respondent. Thus, the emotions of injustice can be associated with feelings of helplessness.

24.2.3.5 Rumination

As we argued earlier, the emotions of injustice can create a psychological and physiological stranglehold over the individual. We add here that rumination not only affects appraisal, but may affect anger directly. That is, rumination may help the emotions of injustice endure over time, sometimes for days, even weeks and months, if not longer. For example, Matthews (1988) recounts an example of an individual who describes his obsession as letting the harmdoer “live inside his head rent free” for years. As Rusting and Nolen-Hoeksema (1998) show, self-focused rumination can maintain or

increase anger. It may be that rumination sustains and prolongs anger, reactivating the emotion every time the victim thinks about the provocation (Chapter 22 by Potegal, this book). For instance, in a longitudinal study on consumers who had suffered service failures for which they consider getting even, Gregoire and Tripp (2007) found that anger had a half-life of more than 2 weeks for those low in trait anger, and for those high in trait anger, the anger sustained out to the last wave of the study (i.e., 8 weeks later). Interestingly, the authors may have helped consumers sustain their anger by repeatedly asking them every 2 weeks if they were still angry. Essentially, these consumers were primed to ruminate, which may have sustained their anger.

24.2.4 Coping Responses

So how do victims cope with the anger? We have investigated two categories of coping responses – cognitive and behavioral. Cognitive coping strategies involve doing nothing behaviorally to resolve or escalate the conflict, whereas behavioral responses are proactive, deliberate changes in actions toward the harmdoer that have the goal of restoring justice or restoring the relationship. Cognitive strategies we have examined are as follows: justifying doing nothing, fantasizing, and forgiveness. Behavioral strategies we have examined are avoidance, revenge, and reconciliation.

24.2.4.1 Justification

One cognitive response is rationalization for doing nothing – that is, not engage in revenge. The explanations for inaction, however, vary. One explanation is *self-resignation*, in which the person just “gives up” and does not think any act of revenge would be effective. A second explanation is that some people do nothing because of a lack of creativity. That is, they simply *cannot invent anything to do*. A third explanation is that many victims simply believe that workplace revenge is always morally wrong. Tripp and Bies (1997) identified several moral judgments that some people make regarding workplace revenge. Many people judge revenge as “unprofessional” or “illegitimate” in that (a) it violates workplace and community norms that, in their opinion, must be upheld or (b) revenge, in their view, never advances the organization’s interests or (c) revenge is an emotional behavior, and workplace behaviors should only follow logical choices. Finally, some victims simply wish to *avoid “grudge costs.”* After interviewing many politicians, Matthews (1988) concluded that getting even costs the avenger so much that the vengeance never exceeds the opportunity costs required to plot and execute it effectively. It is simply better either to “forgive and forget” or to let enough time pass for one to cool off, waiting until a low-cost revenge opportunity emerges that one can rationally execute. As one of Matthews’ subjects quipped, “there’s no point living your life looking in a rearview mirror.” For him, the psychological costs of holding a grudge were too high. One respondent in our data reported spending more time worrying about what might happen than she did enjoying evening the score.

24.2.4.2 Fantasizing

Revenge fantasies were a frequent response in our early, qualitative data (e.g., Bies & Tripp, 1996, cf. Chapter 22 by Potegal, this book). Victims reported vivid, and often violent, dream scenarios, where the victim would “get even” with the harmdoer. Such dreams were filled with rich detail, so much so that one could almost “feel” the pain inflicted by the victim in the act of revenge. In one person’s fantasy she kidnapped her boss, tied him up with duct tape, poured honey all over him, and released bees. This person reported that she would never actually attempt such an action, but just

fantasizing about it relieved much of the stress the injustice had caused. What is interesting is that victims can have revenge fantasies in such elaborate, vivid detail, but with no intention of acting on those feelings.

Some victims went beyond fantasizing and engaged in *plotting* their revenge. Plotting involves the imagining of punishments one could inflict on the harmdoer and may include the scouting for related opportunities, the planning of supplies, and the recruiting of allies needed to carry out a plot. The main difference between plotting and fantasizing is that, in plotting, the victims believe they might actually carry out the plot (i.e., live out the fantasy). Sometimes plotting can be such an obsession that it occurs over years. Matthews (1988) describes several politicians who spent years plotting their revenges.

24.2.4.3 Forgiveness

We define forgiveness as “the *internal* act of relinquishing anger and resentment toward the offender” (Tripp et al., 2007). Forgiveness is an intrapersonal response, where the victim chooses not to retaliate, but instead to feel compassion instead of anger (Enright & the Human Development Study Group, 1991). As one executive stated, “I just stopped feeling angry after I realized how messed up he was as a person” (Bies, 2006).

24.2.4.4 Avoidance

In some cases, victims merely avoid contact with the harmdoer as much as possible. Rather than approach the harmdoer, either cooperatively (e.g., reconciliation) or aggressively (e.g., revenge), the victim retreats from interacting with the harmdoer ever again. The victim finds such interactions unpleasant and aversive, perhaps out of fear of future offenses or perhaps merely out of disgust for the past offense. This type of response leads people to be hypervigilant about the harmdoer’s schedule and presence, so as to avoid the person (Bies & Tripp, 1996).

24.2.4.5 Revenge

We define revenge as “an action in response to some perceived harm or wrongdoing by another party that is intended to inflict damage, injury, discomfort, or punishment on the party judged responsible” (Aquino, Tripp, & Bies, 2001, p. 53). The enactment of revenge can take many forms, depending on the situation and the objective (Bies & Tripp, 1996, 1998; Skarlicki & Folger, 1997; Tripp & Bies, 1997). For example, some forms of revenge resembled inequity reduction responses. For instance, victims might *withhold effort or work* (Bies & Tripp, 1996; Tripp & Bies, 1997), such as deliberately not supporting the harmdoer when support is needed or intentionally turning in poor work performance. Other people sometimes *transfer out of the job or department*, as the ultimate act of withholding support and friendship. In all these acts, the benefit the harmdoer receives from the avenger is reduced or eliminated, thus restoring equity in the relationship.

In other cases, the act of revenge may focus on damaging the other’s reputation or status. In Bies and Tripp (1996) and Tripp and Bies (1997), we found such motives in the following types of revenge: public complaints designed to humiliate another person, public demands for apologies that are intended to embarrass the harmdoer, “badmouthing” the harmdoer, whistle-blowing, and litigation. While whistle-blowing or litigation may not always be intended to harm the harmdoer – that is, it may be intended to stop the wrongdoing, perhaps even in the hopes of reforming the harmdoer or organization (as with pollution or unsafe practices) – our research finds a retributive motivation to “get even” with the harmdoer (Bies & Tripp, 1996).

24.2.4.6 Reconciliation

Aquino et al. (2001) define reconciliation as an effort by the victim to extend acts of goodwill toward the offender in the hope of restoring the relationship (McCullough, Worthington, & Rachal, 1997, 1998). It differs from forgiveness because while forgiveness is intrapersonal, reconciliation is interpersonal. Thus, it is possible to have forgiveness without reconciliation and vice versa. For example, it is possible to overcome negative emotions without hoping to, or even wanting to, restore a relationship with the offender. Conversely, a victim may attempt reconciliation even while still strongly feeling anger or resentment. This might occur if the victim finds it expedient or beneficial to maintain a relationship. In the world of politics, officials may reconcile for specific pieces of legislation (Matthews, 1988) or, in the corporate context, department heads that despise each other will form an alliance to secure resources that would benefit both departments (Bies, 2006).

24.2.5 Moderator Variables

Ours and others’ research shows that the greater the anger, the more vengeful the response. Of course, not all equally angry employees choose the same response: some are more vindictive and volatile than others. What predicts which response they will choose? What other factors may channel their anger into more cooperative or more vengeful responses? At least three classes of factors determine the choice: the victim’s power in the organization, the procedural justice climate of the organization, and the victim’s personality traits.

24.2.5.1 Power

Whether victims get even or not is affected by the power they have. Kim, Smith, and Brigham (1998) showed in an experiment that victims retaliate more often when they have more power than their harmdoers-cum-targets, and that victims retaliate less often when the victims have less power. Aquino et al. (2001, 2006) replicated this same, “relative” power effect in the field study of a government organization. However, they also found that, independent of the harmdoer’s power, the victim’s status in the organization (i.e., where the victim lies in the organizational chart) also affected the likelihood of getting even, but in the opposite direction. That is, the higher placed the victim is in the organization hierarchy, the *less* likely the victim is to get even.

24.2.5.2 Procedural Justice Climate

The fairness of the organization matters to victims. Specifically, the fairer the victims perceive an organization’s procedures to be, the less likely the victim is to get even (Aquino et al., 2001, 2006). Fair procedures are those that, for instance, are applied consistently, allow employees input, avoid conflicts of interest, can be appealed, and generally are ethical (Leventhal, 1980). When procedures are fair, victims believe they can let “the system” handle the offenses committed by harmdoers because the victims believe the system will prosecute the harmdoers and ensure that justice is served. However, when procedures are unfair, then victims believe that if any justice is to be served, they will have to do it themselves by “taking the law into their own hands.” This effect of procedural justice climate is especially pronounced when the victims have less power than their harmdoers – i.e., when they could most use the help from the organization.

24.2.5.3 Personality Traits

Recent research on revenge is showing that traits (at least the traits examined so far) play a *minor* role. In several studies (Skarlicki, Folger, & Tesluk, 1999; Aquino et al., 2001; Tripp et al., 2002), revenge behaviors and attitudes toward revenge behavior have been measured, and the traits (e.g., negative affectivity) have not explained more than 10% of the variance. For instance, in Tripp et al. (2002), situational variables (i.e., perceived consequences of harm) explained 23.4% of the variance in approval of employee-on-employee revenge whereas traits (i.e., age, gender, and belief in the norm of negative reciprocity) explained only 10.3% of the variance.

One major exception to this research is a study by Douglas and Martinko (2001). In a survey of transportation workers and public school employees, Douglas and Martinko found that 62% of the variance in workplace aggression could be explained by the trait variables of trait anger, attitude toward revenge, low self-control, attribution style, and previous exposure to aggressive cultures. However, typical results show traits having a significant role, but rarely larger than the role played by situational traits. A recent meta-analysis of 57 workplace aggression studies (Herschovis et al., 2007) compared situational variables (i.e., distributive justice, procedural justice, interpersonal conflict, job dissatisfaction, and situational constraints) to trait variables (i.e., trait anger, negative affectivity, and gender). In general, the situational variables had slightly larger corrected correlations than did the trait variables, and no variable had corrected correlation larger than 0.41. In terms of significant trait variables, two traits perhaps most examined are trait anger and negative affectivity. In the Herschovis et al. meta-analysis, trait anger was the better predictor of workplace aggression. Future studies on revenge should examine these traits.

24.3 Sense-Making Gone *Really* Bad: When Revenge Causes Feuds

As we have argued, victims contemplating revenge insist on making sense out of their harms, but they are not very accurate at it. They are lousy detectives. Thus, revenge is not only *vigilante* justice (Jacoby, 1983; Tripp et al., 2007), but also *sloppy* justice. This is one reason why vigilantism gets a deservedly bad reputation. Another reason, which we now turn to, is that revenge can escalate a one-shot conflict into a feud. It does so, in part, because of another bias – biased punctuation of conflict history – that stems from the previous biases.

24.3.1 Biased Punctuation of Conflict History

This bias refers to a tendency for individuals to construe the history of conflict with others in a self-serving and provocative fashion (Kramer, 1995). Baumeister, Stillwell, and Wotman (1990) showed just how self-serving such construals can be. In their study, they asked subjects to describe a time when they were angered by someone and a time when they angered someone else. Baumeister et al. found that when subjects were angered (i.e., when they were the victims) they found the provocations arbitrary, gratuitous, or incomprehensible. However, when subjects reflected on times they angered someone else (i.e., when they were the perpetrators), they found their provocations to be justified, meaningful, and comprehensible.

The biased punctuation of conflict describes a specific self-serving construal. In particular, in a two-party conflict, each party believes the other party “started it,” and that the other party is responsible for each escalation of the conflict.

To better illustrate biased punctuation, consider the following example of a protracted and iterated conflict. In this example, two individuals are feuding – an employee, Eric, and his manager, Melinda. On Monday, Eric ignores Melinda’s most recent request. Tuesday, Melinda reprimands Eric for poor performance on a recent task, publicly, embarrassing him. Wednesday, he actively avoids her, knowing that she needs his help on task that day. Thursday, she lets Eric know that she has recorded in Eric’s file her impression of his “lazy” work habits that week and will discuss it formally at performance review. Friday, Eric badmouths Melinda around the office. The following Monday, Melinda hears of the badmouthing and assigns Eric an unpleasant task. . . . And so on the conflict goes. Table 24.1 summarizes the feud.

Table 24.1 Biased punctuation of feud history

	<i>Unbiased, Unpunctuated Reality</i>											
Action by	E	M	E	M	E	M	E	M	E	M
Time	$t_1 - t_{10}$	t_{11}	t_{12}	t_{13}	t_{14}	t_{15}	t_{16}	t_{17}	t_{18}	t_{19}	t_{20}	$t_{21} - t_n$

	<i>Biased Reality: Eric’s Punctuation</i>											
Action by	E	M	E	M	E	M	E	M	E	M	
Time			t_1	t_2	t_3	t_4	t_5	t_6	t_7	t_8	t_9	$t_{10} - t_n$

	<i>Biased Reality: Melinda’s Punctuation</i>											
Action by	E	M	E	M	E	M	E	M	E	M	
Time				t_1	t_2	t_3	t_4	t_5	t_6	t_7	t_8	$t_9 - t_n$

If we were to ask Eric and Melinda separately to explain the cause of the feud, and each were punctuating the conflict with a self-serving bias, we would expect each person to blame the other for the conflict. Specifically, Eric the employee, E, would reinterpret the history of the feud with his manager, Melinda, M, as a sequence of exchanges M-E, M-E, M-E, M-E, in which the initial hostile or aggressive move was made by M. That is, on Tuesday, Melinda “started it” by reprimanding him. So, on Wednesday, Eric withholds help. Then on Thursday – totally unprovoked (because the score was even by Wednesday evening) – Melinda makes a critical recording in Eric’s performance file. Thus, on Friday, Eric gets even by badmouthing Melinda. Then, on Monday, totally unprovoked again (!) (because the score was even after Eric’s last action), Melinda hurts Eric by assigning an unpleasant task. In short, from Eric’s perspective, all of his reactions are legitimate and proportionate responses to malicious, provocative acts by Melinda.

However, Melinda may punctuate the same history of interaction between them as E-M, E-M, E-M, E-M, in which the roles of “offender” and “responder” are reversed. For her, their conflict began on the first Monday, when he ignored her request. It was on Tuesday, when she reprimanded him for poor performance the week before, that the score was even. Thus, when Eric avoided her on Wednesday, that action struck Melinda as unprovoked, because the score was even the previous

afternoon. Thus, from Melinda's perspective, each of Eric's actions is an unprovoked misbehavior or attack, while each of her actions is a legitimate and proportionate response.

In terms of its role in the etiology of revenge behavior, the importance of biased punctuation of conflict is threefold. First, it contributes to self-justificatory motives. As Frank (1987) perceptively notes in this regard, construing the history of conflict in this way can be used to justify the claim that one needs "to defend against a powerful and evil enemy, *thereby shifting responsibility for one's own aggressive actions to the opponent*" (p. 340, emphasis added). Bies and Tripp (1996) found a similar justification for revenge behavior by avengers, who viewed their actions as "morally right" and "in service of justice."

Second, biased punctuation of conflict tends to generate self-fulfilling patterns of action–reaction between the parties as each tries to restore balance to the relationship by "evening" the score (Kahn & Kramer, 1990). In other words, both sides view their own actions as purely defensive behaviors made in response to the other's unwarranted actions.

Third, it suggests how dangerous it can be to respond "tit for tat" to the other party in a futile attempt to "even the score." Clearly, because of the biases, both parties are not using the same score card, and thus they will never agree that at some point in time the score is even. One party will always be "behind," and thus looking to even the score through further aggression. We suspect that such feuding can lead to hatred.

24.4 Conclusion

In a nutshell, there is clear and consistent empirical evidence that revenge is motivated by a righteous anger, thus providing revenge its own moral imperative. For, revenge is, in many cases, a response to a perceived injustice. Second, revenge is most often intended to restore justice. For instance, while engaging in revenge, people reported their strong belief that they were "doing the right thing" and that they were "doing justice." Third, while the act of revenge may have served self-interest, it often serves other interests, and it is usually justified in moral terms. The justice rationality can be a powerful motivation and justification for revenge.

In assessing the morality of revenge as an observer, one must use not just the organization's interests. One must look also at revenge through the eyes of the avenger and "innocent bystanders" in assessing the morality of revenge. In other words, the morality of revenge must be evaluated in terms of three different stakeholders who may be affected: the *avenger*, the *perpetrator*, and *bystanders* (Bies & Tripp, 1998). There may be times where revenge does more good than harm, even if the good is to the employees and the harm is to (some members of) the upper management. Certainly, that is the way the avengers often view it – and justify it.

If we, as researchers, wish to understand when and why people in organizations become avengers and to understand what form and level of vengeance they seek, then we must understand the avengers' perspectives. This means that we must consider goals and viewpoints other than those prescribed by top management. Furthermore, we must also assume that avengers are rational and moral beings: they are goal directed, respond to their environment, often coolly calculate the costs and benefits of their actions, and justify their actions. They are not necessarily random, "crazed," impetuous, petulant, or otherwise stupid or evil, as portrayed by the popular stereotypes of vengeance seekers and their acts of revenge (Barreca, 1995; Jacoby, 1983).¹

¹We owe a debt of gratitude to Rod Kramer for his contribution in stimulating and enriching our analysis of revenge.

References

- Adams, S. (1965). Inequity in social exchange. In L. Berkowitz (Ed.), *Advances in experimental social psychology* (Vol 2, pp. 267–299). New York: Academic Press.
- Aquino, K., Tripp, T. M., & Bies, R. J. (2001). How employees respond to personal offense: The effects of blame attribution, victim status, and offender status on revenge and reconciliation in the workplace. *Journal of Applied Psychology, 86*, 52–59.
- Aquino, K., Tripp, T. M., & Bies, R. J. (2006). Getting even or moving on? Status variables and procedural justice as predictors of revenge, forgiveness, and reconciliation in organizations. *Journal of Applied Psychology, 91*, 653–668.
- Asch, S. E. (1951). Effects of group pressure upon the modification and distortion of judgment. In H. Guetzkow (Ed.), *Groups, leadership and men*. Pittsburgh, PA: Carengie University Press.
- Averill, J. R. (1982). *Anger and aggression: An essay on emotion*. New York: Springer-Verlag.
- Baron, R. A. (1988). Negative effects of destructive criticism: Impact on conflict, self-efficacy, and task performance. *Journal of Applied Psychology, 73*, 199–207.
- Baron, R. A. (1990). Countering the effects of destructive criticism: The relative efficacy of four interventions. *Journal of Applied Psychology, 75*, 235–245.
- Barreca, R. (1995). *Sweet revenge: The wicked delights of getting even*. New York: Harmony Books.
- Baumeister, R. F., Stillwell, A., & Wotman, S. R. (1990). Victim and perpetrator accounts of interpersonal conflict: Autobiographical narratives about anger. *Journal of Personality and Social Psychology, 59*, 994–1005.
- Bies, R. J. (1987). The predicament of injustice: The management of moral outrage. In L. L. Cummings and B. M. Staw (Eds.), *Research in organizational behavior* (Vol. 9, pp. 289–319). Greenwich, CT: JAI Press.
- Bies, R. J. (2001). Interactional (in)justice: The sacred and the profane. In J. Greenberg & R. Cropanzano (Eds.), *Advances in organizational behavior* (pp. 89–118). San Francisco: The New Lexington Press.
- Bies, R. J. (2006). *Peering into the soul of discontent: Revenge and forgiveness in the workplace*. Unpublished manuscript.
- Bies, R. J. & Tripp, T. M. (1996). Beyond distrust: “Getting even” and the need for revenge. In R. M. Kramer & T. Tyler (Eds.), *Trust and organizations* (pp. 246–260). Thousand Oaks, CA: Sage.
- Bies, R. J. & Tripp, T. M. (1998). The many faces of revenge: The good, the bad, and the ugly. In R. W. Griffin, A. O’Leary-Kelly, & J. Collins (Eds.), *Dysfunctional behavior in organizations, Vol. 1: Violent behaviors in organizations* (Vol. 23, pp. 49–68). Greenwich, CT: JAI Press.
- Bies, R. J. & Tripp, T. M. (2001). A passion for justice: The rationality and morality of revenge. In R. Cropanzano (Ed.), *Justice in the workplace* (Vol. II). Mahwah, NJ: Lawrence Erlbaum Associates.
- Bies, R. J. & Tripp, T. M. (2002). Hot flashes, open wounds: Injustice and the tyranny of its emotions. In S. Gilliland, D. Steiner, and D. Skarlicki (Eds.), *Emerging perspectives on managing organizational justice* (pp. 203–223). Greenwich, CT: Information Age.
- Bies, R. J. & Tripp, T. M. (2004). The study of revenge in the workplace: Conceptual, ideological, and empirical issues. In S. Fox & P. Spector (Eds.), *Counterproductive workplace behavior: An integration of both actor and recipient perspectives on causes and consequences* (pp. 65–82). Washington, DC: American Psychological Association.
- Bies, R. J., Tripp, T. M., & Kramer, R. M. (1997). At the breaking point: Cognitive and social dynamics of revenge in organizations. In R. A. Giacalone & J. Greenberg (Eds.), *Anti social behavior in organizations* (pp. 18–36). Thousand Oaks, CA: Sage.
- Bies, R. J. & Tyler, T. R. (1993). The litigation mentality in organizations: A test of alternative psychological explanations. *Organization Science, 4*, 352–366.
- Buss, A. H. (1962). *The psychology of aggression*. New York: Wiley.
- Cahn, E. (1949). *The sense of injustice*. New York: New York University Press.
- Cohen, R. L. (1982). Perceiving justice: An attributional perspective. In J. Greenberg, & R. L. Cohen (Eds.), *Equity and justice in social behavior* (pp. 119–160). New York: Academic Press.
- Colby, K. M. (1981). Modeling a paranoid mind. *Behavioral and Brain Sciences, 4*, 515–560.
- Crosby, F. (1976). A model of egoistic relative deprivation. *Psychological Review, 83*, 85–113.
- Crossley, C. D. (2006). The role of offender malice and greed in behavioral reactions to social undermining. In K. M. Weaver (Ed.), *Proceedings of the Sixty-fifth Annual Meeting of the Academy of Management (CD)*, Atlanta, GA, ISSN 1543-8643.
- Deutsch, M. (1975). Equity, equality, and need: What determines which value will be used as the basis of distributive justice? *Journal of Social Issues, 31*, 137–149.
- Douglas, S. C. & Martinko, M. J. (2001). Exploring the role of individual differences in the prediction of workplace aggression. *Journal of Applied Psychology, 86*, 547–559.

- Enright, R. D., & The Human Development Study Group. (1991). The moral development of forgiveness. In W. Kurtines & J. Gewirtz (Eds.), *Handbook of moral behavior and development* (Vol. 1, pp. 123–152). Hillsdale, NJ: Erlbaum.
- Feinberg, J. (1974). Noncomparative justice. *The Philosophical Review*, 83, 297–338.
- Felstiner, W. L. F., Abel, R. F., & Sarat, A. (1980–1981). The emergence and transformation of disputes: Naming, blaming, claiming. *Law and Society Review*, 15, 631–654.
- Ferguson, T. J., & Rule, B. G. (1983). An attributional perspective on anger and aggression. In R. G. Geen & E. I. Donnerstein (Eds.), *Aggression: Theoretical and empirical reviews* (pp. 41–74). New York: Academic Press.
- Frank, J. D. (1987). The drive for power and the nuclear arms race. *American Psychologist*, 42, 337–344.
- Garrett, J., & Libby, W. (1973). Role of intentionality in mediating responses to inequity in the dyad. *Journal of Personality and Social Psychology*, 28, 21–27.
- Goldman, B. (2003). The application of referent cognitions theory to legal-claiming by terminated workers: The role of organizational justice and anger. *Journal of Management*, 29, 705–728.
- Greenberg, J. (1990). Employee theft as a reaction to underpayment inequity: The hidden costs of pay cuts. *Journal of Applied Psychology*, 75, 561–568.
- Gregoire, Y. & Tripp, T. M. (2007). *Consumer revenge*. Working paper.
- Hastorf, A. & Cantril, H. (1954). They saw a game: A case study. *Journal of Abnormal and Social Psychology*, 49, 129–134.
- Herscovis, M. S., Turner, N., Barling, J., Arnold, K. A., Dupre, K. E., et al. (2007). Predicting workplace aggression: A meta-analysis. *Journal of Applied Psychology*, 92, 228–238.
- Hogan, R. & Emler, N. P. (1981). Retributive justice. In M. J. Lerner and S. C. Lerner (Eds.), *The justice motive in social behavior* (pp. 125–143). New York: Plenum.
- Hornstein, H. A. (1996). *Brutal bosses and their prey*. New York: Riverhead Books.
- Jacoby, S. (1983). *Wild justice: The evolution of revenge*. New York: Harper & Row.
- Janis, I. L. (1983). *Groupthink*, (2nd ed.). Boston: Mifflin.
- Jones, E. E., Kanouse, D. E., Kelley, H. H., Nisbett, R. E., Valines, S., & Weiner, B. (1972). *Attribution: Perceiving the causes of behavior*. Morristown, NJ: General Learning Press.
- Jones, E. E. & Nisbett, R. E. (1972). The actor and the observer: Divergent perceptions of the causes of behavior. In E. E. Jones, D. E. Kanouse, H. H. Kelley, R. E. Nisbett, S. Valins, & B. Weiner (Eds.), *Attribution: Perceiving the causes of behavior* (pp. 79–94). Morristown, NJ: General Learning Press.
- Kahn, R. L. & Kramer, R. M. (1990). Untying the knot: De-escalatory processes in international conflict. In R. L. Kahn, M. N. Zald (Eds.), *Organizations and nation-states: New perspectives on conflict and cooperation*. San Francisco: Jossey-Bass.
- Kelley, H. H. (1972). Attribution in social interaction. In E. E. Jones, D. E. Kanouse, H. H. Kelley, R. E. Nisbett, S. Valins, & B. Weiner (Eds.), *Attribution: Perceiving the causes of behavior* (pp. 1–26). Morristown, NJ: General Learning Press.
- Kim, S. H., Smith, R. H., & Brigham, N. L. (1998). Effects of power imbalance and the presence of third parties on reactions to harm: Upward and downward revenge. *Personality and Social Psychology Bulletin*, 24, 353–361.
- Kramer, R. M. (1994). The sinister attribution error. *Motivation and Emotion*, 18, 199–231.
- Kramer, R. M. (1995). The distorted view from the top: Power, paranoia, and distrust in organizations. In R. Bies, R. Lewicki, and B. Sheppard (Eds.), *Research on Negotiations* (Vol. 5). Greenwich, CT: JAI Press.
- Kramer, R. M. (2001). Organizational paranoia: Origins and dynamics. In B. Staw & R. I. Sutton (Eds.), *Research in organizational behavior*, 23, 1–42.
- Leventhal, G. S. (1980). What should be done with equity theory? New approaches to the study of fairness in social relationships. In K. Gergen, M. Greenberg, & R. Willis (Eds.), *Social exchange: Advances in theory and research* (pp. 27–55). New York: Plenum Press.
- Leventhal, G.S., Weiss, T. & Long, G. Equity, reciprocity, and reallocating rewards in the dyad. *Journal of Personality and Social Psychology*, 13, 300–305.
- Lyubomirsky, S. & Nolen-Hoeksema, S. (1993). Self-perpetuating properties of dysphoric rumination. *Journal of Personality and Social Psychology*, 65, 339–349.
- Matthews, C. (1988). *Hardball: How politics is played – told by one who knows the game*. New York: Summit Books.
- McCullough, M. E., Rachal, K. C., Sandage, S. J., Worthington, E. L., Brown, S. W., & Hight, T. L. (1998). Interpersonal forgiving in close relationships: Theoretical elaboration and measurement. *Journal of Personality and Social Psychology*, 75, 1586–1603.
- McCullough, M. E., Worthington, E. L., & Rachal, K. C. (1997). Interpersonal forgiving in close relationships. *Journal of Personality and Social Psychology*, 73, 321–336.

- McLean-Parks, J. M. (1997). The fourth arm of justice: The art and science of revenge. In R. J. Lewicki, R. J. Bies, & B. H. Sheppard (Eds.), *Research on negotiation in organizations* (Vol. 6, pp. 113–144). Greenwich, CT: JAI Press.
- Mikula, G. (1986). The experience of injustice: Toward a better understanding of its phenomenology. In H. W. Bierhoff, R. L. Cohen, & J. Greenberg (Eds.), *Justice in interpersonal relations* (pp. 103–123). New York: Plenum Press.
- Mikula, G., Petri, B., & Tanzer, N. (1990). What people regard as just and unjust: Types and structures of everyday experiences of injustice. *European Journal of Social Psychology*, 20, 133–149.
- Miller, D. T. (2001). Disrespect and the experience of injustice. *Annual Review of Psychology*, 52, 527–553.
- Miller, D. & Vidmar, N. (1981). The social psychology of punishment reactions. In S. Lerner & M. Lerner (Eds.), *The justice motive in social behavior* (pp. 145–167). New York: Plenum Press.
- Mishra, A. K. & Spreitzer, G. M. (1998). Explaining how survivors respond to downsizing: The roles of trust, empowerment, justice, and work redesign. *Academy of Management Review*, 23, 567–588.
- Morrill, C. (1992). Vengeance among executives. *Virginia Review of Sociology*, 1, 51–76.
- Neuman, J. H. & Baron, R. A. (1997). Aggression in the workplace. In R. A. Giacalone and J. Greenberg (Eds.), *Antisocial behavior in organizations* (pp. 37–67). Thousand Oaks, CA: Sage Publications.
- Pfeffer, J. (1992). *Managing with power*. Cambridge, MA: Harvard Business School.
- Pomfret, J. (1995, December 18). Atrocities leave thirst for vengeance in Balkans. *The Washington Post*, A1.
- Pyszczynski, T. & Greenberg, J. (1987). Self-regulatory perseveration and the depressive self focusing style: A self-awareness theory of reactive depression. *Psychological Bulletin*, 102, 122–138.
- Robinson, S. L. & Bennett, R. J. (1995). A typology of deviant workplace behaviors: A multidimensional scaling study. *Academy of Management Journal*, 38, 555–572.
- Ross, L. & Nisbett, R. E. (1992). *The person and the situation*. New York: McGraw-Hill.
- Rusting, C. L. & Nolen-Hoeksema, S. (1998). Regulating responses to anger: Effects of rumination and distraction on angry mood. *Journal of Personality and Social Psychology*, 74, 790–803.
- Salancik, G. R. & Pfeffer, J. (1978). A social information processing approach to job attitudes and task design. *Administrative Science Quarterly*, 23, 224–253.
- Sheppard, B. H., Lewicki, R. J., & Minton, J. W. (1992). *Organizational justice: The search for fairness in the workplace*. New York: Lexington Books.
- Skarlicki, D. P. & Folger, R. (1997). Retaliation in the workplace: The roles of distributive, procedural, and interactional justice. *Journal of Applied Psychology*, 82, 434–443.
- Skarlicki, D. P., Folger, R., & Tesluk, P. (1999). Personality as a moderator in the relationship between fairness and retaliation. *Academy of Management Journal*, 42, 100–108.
- Solomon, R. C. (1990). *A passion for justice: Emotions and the origins of the social contract*. Reading, MA: Addison-Wesley.
- Thomas, K. W. (1976). Conflict and conflict management. In M. D. Dunnette (Ed.), *The handbook of industrial and organizational psychology*. Chicago, IL: Rand McNally.
- Thomas, K. W. & Pondy, L. R. (1977). Toward an “intent” model of conflict management among principal parties. *Human Relations*, 30, 1089–1102.
- Tripp, T. M. & Bies, R. J. (1997). What’s good about revenge? In R. J. Lewicki, R. J. Bies, and B. H. Sheppard (Eds.), *Research on negotiation in organizations* (Vol. 6, pp. 145–160). Greenwich, CT: JAI Press.
- Tripp, T. M., Bies, R. J., & Aquino, K. (2002). Poetic justice or petty jealousy? The aesthetics of revenge. *Organizational Behavior and Human Decision Processes*, 89, 966–984.
- Tripp, T. M., Bies, R. J., & Aquino, K. (2007). A vigilante model of justice: Revenge, reconciliation, forgiveness, and avoidance. *Social Justice Research*, 19, 10–34.
- Utne, M. K. & Kidd, R. F. (1980). Equity and attribution. In G. Mikula (Ed.), *Justice and social interaction* (pp. 63–93). New York: Springer-Verlag.
- Wason, P. (1960). On the failure to eliminate hypotheses in a conceptual task. *Quarterly Journal of Experimental Psychology*, 12, 129–140.
- Weick, K. E. (1995). *Sensemaking in organizations*. Thousand Oaks, CA: Sage.
- Wilson, T. D. & Kraft, D. (1993). Why do I love thee? Effects of repeated introspections about a dating relationship on attitudes towards the relationship. *Personality and Social Psychology Bulletin*, 19, 409–418.
- Wong, P. T. & Weiner, B. (1981). When people ask “why” questions, and the heuristics of attributional search. *Journal of Personality and Social Psychology*, 40, 650–663.

Part VIII
Clinical Aspects of Anger

Chapter 25

Anger/Hostility and Cardiovascular Disease

Janice E. Williams

Abstract Cardiovascular disease (CVD) comprises diseases of the heart and the circulatory system, of which coronary heart disease (CHD) and stroke are major clinical end points. CVD is the leading cause of death in industrialized nations and, owing to its rapid acceleration in developing countries, is projected to become the number one killer worldwide. The established CVD risk factors are age, physical inactivity, diabetes mellitus, hypertension, elevated triglyceride levels, elevated low-density lipoprotein cholesterol levels, and cigarette smoking. Concern for the CHD morbidity and mortality burden and interest in effective prevention have stimulated debate over the degree to which new cases can be attributed to the traditional risk factors. Several studies have shown that after accounting for the traditional risk factors, unexplained variance in CHD remains. There has been growing interest in the influence of psychological factors in CVD. This chapter reviews primarily prospective, population-based studies on the relationship between anger/hostility and CVD. The results from these investigations confirm that trait anger/chronic hostility, anger expression, and acute anger episodes have positive predictive value for CVD – including new or recurrent events or atherosclerosis.

Cardiovascular disease (CVD) comprises diseases of the heart and the circulatory system, of which coronary heart disease (CHD) and stroke are major clinical endpoints. CVD is the leading cause of death in industrialized nations and, owing to its rapid acceleration in developing countries, is projected to become the number one killer worldwide (Levenson, Skerrett, & Gaziano, 2002). The established CVD risk factors are: age, physical inactivity, diabetes mellitus, hypertension, elevated low density lipoprotein-cholesterol levels, elevated triglyceride levels, and cigarette smoking. Within the spectrum of CVD outcomes, CHD is the single leading cause of death. Concern for the CHD morbidity and mortality burden and interest in effective prevention have stimulated debate over the degree to which new cases can be attributed to the traditional risk factors. Several studies have shown that, after accounting for the traditional risk factors, there is still unexplained variance in CHD, although the precise amount of the residual remains unclear (Beaglehole & Magnus, 2002).

Negative affect is emerging as an important risk factor for CVD, potentially linked to this disease by indirect, behavioral and/or direct, pathophysiological mechanisms. Anger and its close kin, hostility, have been widely investigated for their impact on CVD. Anger and hostility are closely related constructs, yet there are some important distinctions between them. Anger is the core emotional

J.E. Williams (✉)
Program in Public Health, LaGrange College, LaGrange, GA, USA
e-mail: jwill22@bellsouth.net

aspect of hostility; however, hostility consists of additional behavioral (e.g., aggression) and cognitive (e.g., cynicism, mistrust, a negative predisposition toward others, resentfulness, suspiciousness) components. The degree to which a person is prone to experience anger is referred to as trait anger (Chapter 23 by C.D. Spielberger and E.C. Reheiser, this book). People who have high trait anger experience irritation, fury, and rage more frequently and for longer periods of time compared to people who have low trait anger.

Current studies on anger/hostility and CHD have their historical roots in Friedman and Rosenman's (Friedman & Rosenman, 1959) seminal work on the Type A behavior pattern and coronary disease. In their classic 9-year follow-up study, these authors reported that men who exhibited a behavioral pattern characterized by ambitiousness, competitive drive, time urgency, impatience, hostility, and aggressiveness (Type A) compared to their Type B (relaxed, patient) counterparts were at increased risk for myocardial infarction (MI). Subsequent research has revealed that, of the global Type A behavior pattern attributes, negative affect, namely anger/hostility, has the most deleterious consequences for CHD (Matthews, Glass, Rosenman, & Bortner, 1977).

Despite some reported non-replications (Hearn, Murray, & Luepker, 1989; McCranie, Watkins, Brandsma, & Sisson, 1986), the weight of the research evidence favors a significant positive association between anger/hostility and major CHD events. Prospective studies on this relationship have differed in design features such as the measurement of anger/hostility, length of follow-up, risk factors and endpoints assessed, cohort size, the racial/ethnic makeup of the participants, and whether participants had preexisting CHD at baseline. A meta-analysis of 45 studies revealed that, on balance, anger/hostility was positively associated with CHD and all-cause mortality, independent of potential biologic and sociodemographic confounders (Miller, Smith, Turner, Guijarro, & Hallet, 1996). Since that analysis, published in 1996, several large, population-based studies have supported this conclusion. There is also evidence that anger/hostility can confer a CHD risk that is comparable to (Kop, 1997) and even greater than that which is conferred by the traditional risk factors (Chaput et al., 2002).

This chapter reviews the relationship between anger/hostility and CVD. It provides an overview of studies (primarily prospective, population based) on the relationship between anger/hostility and new CVD/CHD cases, recurrent CVD/CHD, atherosclerosis, and new stroke cases. It is not intended as an exhaustive summary; rather, it includes selected studies that are illustrative of anger/hostility's role in the initiation and progression of CVD. Also described are the pathogenic mechanisms that are thought to underlie the anger/hostility–CVD association.

25.1 Trait Anger/Chronic Hostility

25.1.1 New CVD/CHD Events

Several long-term follow-up studies have reported a positive association between anger/hostility and new CVD/CHD events among participants who were initially healthy at study enrollment. The Johns Hopkins Precursor Study, begun in 1946, is one of the oldest cohort studies of this type (Chang et al., 2002). Investigators assessed the relationship between anger and premature CVD (events occurring before age 55) and total CVD (events occurring before and after age 55) among 1,055 male medical students at Johns Hopkins University. At the study baseline, participants were assessed for their usual response to stress using the Habits of Nervous Tension Questionnaire. They were followed 32–48 years for the occurrence of CVD events. Men typically responding to stress with (1)

“expressed or concealed anger, (2) “gripe sessions”, and (3) “irritability,” compared to their counterparts who typically responded with less than all three of these behaviors, had relative risks of 3.1, 3.5, and 6.4 for premature CVD, premature CHD, and premature MI, respectively, after adjusting for potential confounders. These effects were not observed for total CVD, nor were they observed in the subgroup of men who had CVD events at age 55 and older, indicating effect modification by age.

Analyses from the Atherosclerosis Risk in Communities (ARIC) Study support a role for trait anger in CHD onset and CHD mortality (Williams et al., 2000). The ARIC Study is a large epidemiologic investigation that consisted of a bi-ethnic cohort of middle-aged men and women residing in the following US communities: suburban Minneapolis, MN; Washington County, MD; Forsyth County, NC; and Jackson, MS. It is one of the few prospective studies of anger/hostility and CHD that has included substantial numbers of women and African-Americans. Across an average of 6 years, trait anger was positively associated with the risk for the combined endpoint of revascularization procedures (coronary artery bypass grafting or percutaneous coronary transluminal angioplasty), acute MI, or cardiac death among the 12,986 participants (45–64 years of age) who were free of CHD at baseline. Anger was assessed using the Spielberger Trait Anger Scale. Participants who scored in the high trait anger range were twice as likely to have a combined CHD event compared to their counterparts who scored in the low range. The association was stronger for “hard” events (e.g., acute MI or cardiac death), for which the risk was nearly three times as great. These effects, adjusted for potential biologic and sociodemographic confounders, were observed among individuals with normal blood pressure levels, but not among those with high levels. No differences by race/ethnicity were found.

When the trait anger subscales (anger temperament and anger reaction) were examined, it was found that a fiery, volatile temperament (anger temperament) was more strongly related to CHD onset than anger aroused in reaction to frustration, criticism, or unfair treatment (anger reaction) (Williams, Nieto, Sanford, & Tyroler, 2001). People with a strong angry temperament are quick to express anger even when there is no obvious cause. An angry temperament is more enduring than reactive anger, since anger temperament, which may have constitutional underpinnings, persists across time and contexts. These results suggest that, of the Spielberger Trait Anger subscales, a chronic antagonistic disposition has more adverse CHD consequences. Again, these effects were observed among normotensive individuals, but not among hypertensives. The differences by hypertensive status may be due to the fact that hypertension alone confers substantial CHD risk. Thus, in a high-risk setting, any anger effects might have been minimized.

Using a case-control study design, the relationship of hostility to CVD was assessed in the Multiple Risk Factor Intervention Trial (MRFIT), another large-scale, population-based investigation (Matthews, Gump, Harris, Haney, & Barefoot, 2004). Participants were 259 men (ages 35–57 at baseline) who were followed for 16 years (7.1 years in the trial and 8.9 years posttrial, on average). Hostility was assessed using The Interpersonal Hostility Assessment Technique – a structured interview. In the follow-up period, men in the high hostility group had a 61% greater likelihood of a fatal CVD event compared to their low-hostile counterparts, adjusting for diastolic blood pressure and cholesterol levels, smoking status, and nonfatal CVD events during the trial. The risk for an event *after* the trial was even greater among high-hostile men who experienced a nonfatal event *during* the trial (compared to their low-hostile counterparts who did not experience a nonfatal event during the trial), among whom an odds ratio of 5.06 was reported.

Prospective analyses have also shown that the anger/hostility–CVD relationship may depend upon the risk factors. Analyses among 2,125 middle-aged men (aged 42–60 years) enrolled in the Kuopio Ischemic Heart Disease Risk Factor Study demonstrated that the hostility–CVD relationship was mediated by CVD risk factors across 9 years of follow-up (Everson et al., 1997). Hostility

was assessed using the Cynical Distrust Scale of the Cook-Medley Hostility Scale. In age-adjusted models, high hostility compared to low hostility, conferred relative risks of 2.3 and 2.7 for MI and cardiovascular mortality, respectively. However, additional adjustment by traditional CVD and psychosocial risk factors substantially attenuated these effects. Chief among those factors were cigarette smoking, physical activity, body mass index, and alcohol use. Structural equation modeling of data from the Swedish Adoption/Twin Study of Aging showed that a positive relationship between hostility and CVD could be explained, at least in part, by the metabolic syndrome (defined as a clustering of CVD risk factors, including abdominal adiposity, and high triglyceride-, high blood pressure-, and high LDL-cholesterol levels) (Nelson, Palmer, & Pedersen, 2004). This analysis was adjusted for cigarette smoking, age, and prevalent CVD. Participants were 1,944 men and women, aged 62 years, on average, at study baseline.

25.1.2 Recurrent CVD/CHD Events

Anger/hostility also has been positively associated with recurrent cardiovascular events. Hostility independently predicted recurrent CHD among 792 postmenopausal women (67 years of age, on average) who were enrolled in the Heart and Estrogen/Progestin Replacement Study (Chaput et al., 2002). At baseline, enrollees had a history of CHD, including MI, coronary artery bypass graft surgery, occlusion of a coronary vessel, or coronary revascularization procedure. An average of 4.1 years later, women in the highest quartile of hostility had an 88% greater risk for the combined endpoint of nonfatal MI or CHD death and were twice as likely to have an MI compared to their counterparts in the lowest quartile. Additionally, across 4 years of follow-up, hostility was reported as an independent risk factor for recurrent CHD (hospitalizations or death) among men with a history of CHD in the Nova Scotia Health Survey. High-hostile men were at twice the risk for recurrent CHD compared to their low-hostile counterparts (Haas et al., 2005). No such associations were observed in the women.

In another report, trait anger predicted recurrent CHD events among 149 men and women who had undergone percutaneous transluminal coronary angioplasty (Mendes de Leon, Kop, de Swart, Bar, & Appels, 1996). Participants, aged 70 years or younger, were followed for 18 months after their procedure. The results showed that high trait anger conferred a statistically significant crude relative risk of 2.09 for subsequent events (MI, cardiac death, coronary artery bypass graft surgery, additional angioplasty, new or progressed coronary lesions, or angina pectoris), owing primarily to the association observed in men. The risk estimate became marginally significant in multivariate analyses. When trait anger and vital exhaustion together were assessed, the multivariate-adjusted risk for a recurrent event was statistically significant and greater than that for trait anger alone. Vital exhaustion is a condition defined as increased irritability, demoralization, and excess fatigue and is often considered to be the result of prolonged stress. In addition, vital exhaustion is frequently observed in the prodromal period of an MI and is positively associated with MI onset (Appels, 1997).

Similarly, potential for hostility and the risk for restenosis of a coronary artery were examined in a smaller sample of 41 men and women, aged 57.8 on average, who also had undergone angioplasty (Goodman, Quigley, Moran, Meilman, & Sherman, 1996). Potential for hostility was assessed using the Type A Structured Interview. The results indicated that participants with high potential for hostility were at more than twice the risk for restenosis compared to their low-hostile counterparts. Hostility scores also were positively correlated with the number of restenosed vessels. Compared to the effects of body mass index, diabetes mellitus, and cigarette smoking status, potential for hostility was the only statistically significant risk factor for restenosis.

25.1.3 Stroke

Compared to CHD, much less evidence on the relationship between anger/hostility and stroke exists. An analysis of ARIC data showed a positive association between trait anger and incident stroke that was modified by age and HDL-cholesterol level (Williams, Nieto, Sanford, Couper, & Tyroler, 2002). After adjusting for several established biologic and sociodemographic risk factors, men and women with high trait anger, aged 60 years or younger, were at more than twice the risk for ischemic stroke and at nearly twice the risk for any stroke (ischemic or hemorrhagic) relative to their low anger and more aged counterparts. Similarly, participants with high trait anger and HDL-cholesterol levels greater than 47 were at two and one-half times the risk for ischemic stroke and at more than twice the risk for any stroke compared to their counterparts with low anger and low HDL-cholesterol levels.

Another important finding pertaining to stroke is evidence of a positive association between anger/hostility and a significant stroke risk factor, atrial fibrillation, in men (Eaker et al., 2004). Data from 3,873 men and women, aged 18–77 years, who were enrolled in the Framingham Offspring Study, demonstrated that across the 10-year follow-up period, trait anger, anger symptoms, and hostility were independently and positively associated with incident atrial fibrillation in men, but not in women. Trait anger also predicted total mortality in men only.

25.1.4 Atherosclerosis

Prospective and cross-sectional analyses indicate a positive association between anger/hostility and indices of subclinical carotid and coronary atherosclerosis – some associations have been independent of the risk factors and others have not. Further, similar to the studies on anger/hostility and CHD, non-replications have been reported (Helmer, Ragland, & Syme, 1991; O'Malley, Jones, Feuerstein, & Taylor, 2000). In the 10-year follow-up period of the Coronary Artery Risk Development In Young Adults (CARDIA) Study, hostility was independently associated with the development of coronary artery calcification in men and women, aged 18–30 years at study entry (Iribarren et al., 2000). Among middle-aged women, trait anger predicted the progression of carotid artery intima-media thickness (IMT) over 3 years of follow-up, although the metabolic syndrome accounted for the relationship (Räikkönen, Matthews, Sutton-Tyrrell, & Kuller, 2004). IMT is a manifestation of subclinical atherosclerosis and is characterized by thickening of the intimal and medial arterial walls.

In cross-sectional analyses, overall trait anger, anger temperament, and anger out were significantly and independently associated with maximum carotid artery IMT levels in untreated hypertensive men, aged 40–70 years (Bleil, McCaffery, Muldoon, Sutton-Tyrrell, & Manuck, 2004). Overall trait anger and anger temperament were marginally related to mean IMT. In addition, anger temperament significantly predicted the presence of atherosclerotic plaque. Further, among African-Americans and whites in the Study of Women's Health Across The Nation, hostility was positively associated with mean and maximum IMT levels in multivariate-adjusted analyses (Everson-Rose et al., 2006). In ARIC, trait anger was positively and independently associated with carotid artery IMT in African-American men (Williams, Couper, Din-Dzietham, Nieto, & Folsom, 2007). In other race/gender groups, the trait anger–carotid IMT association appeared to have been mediated by the traditional CHD risk factors. Also in ARIC, trait anger was examined for its relationship to carotid artery stiffness, a functional property of the artery indicative of subclinical atherosclerosis (Williams, Din-Dzietham, & Szklo, 2006). The results showed a positive association in men, but not in women. In men, the association was nonlinear, but the change in arterial stiffness from low to high trait anger was large and clinically significant.

In sum, studies continue to confirm that trait anger/chronic hostility has positive predictive value for CVD – including new CHD events, recurrent CHD, new stroke events, and atherosclerosis. Like their predecessors, the large prospective studies of incident CHD and stroke have included primarily white males. Some of these investigations confirm earlier reports (Meesters & Smulders, 1994) showing that the influence of trait anger/hostility on CVD diminishes with increasing age. However, there have been few reports of effect modification by gender and race/ethnicity. On the question of whether the trait anger/chronic hostility–CVD association is independent of the risk factors, the findings are still mixed. The differences in populations, sample sizes, lengths of follow-up, risk factors assessed, and differences in the methods used to assess anger/hostility make this issue difficult to resolve.

25.2 Anger Expression and Control

The manner in which people characteristically express anger (e.g., holding in angry feelings [anger in], overtly expressing them [anger out], and degree of effort to control the outward expression and intensity of angry feelings [anger control]) is collectively referred to as anger expression. These characteristics are psychometrically distinct, meaning that any one individual can have any degree of each trait (Chapter 23 by C.D. Spielberger and E.C. Reheiser, this book). Therefore, each characteristic can have its own relationship to CVD.

25.2.1 New CVD/CHD Events

The relationship of anger control to CVD/CHD was examined in the Veterans Administration Normative Aging Study – a prospective cohort of 1,305 men who were aged 21–80 years at study enrollment (Kawachi, Sparrow, Spiro, Vokonas, & Weiss, 1996). Participants were free of CHD at baseline and were assessed for anger using the Anger Content Scale of the Minnesota Multiphasic Personality Inventory (MMPI)-2. They were followed an average of 7 years for evidence of angina pectoris, MI, or fatal CHD. Independent of the established risk factors, the results showed that the CHD risk was 2.66 times as great among men who reported high anger control compared to their low anger control counterparts.

Anger expression and CVD/CHD have also been examined. In an analysis of 2,890 middle-aged men (aged 49–65 years at baseline) enrolled in a study conducted in the Caerphilly, South Wales, Framingham anger items (e.g., anger in, anger out, anger symptoms, and anger discuss) and a suppressed anger scale, derived for use in this analysis, were used to assess the relationship between anger expression and CHD (Gallacher, Yarnell, Sweetnam, Elwood, & Stansfeld, 1999). Participants, including those with and without evidence of ischemia at baseline, were followed for approximately 9 years. Results from this analysis showed a 69% and 57% greater likelihood of a CHD event among men who reported low anger out and high suppressed anger, respectively. These associations were independent of potential physiological, behavioral, and psychosocial confounders. Further, multivariate-adjusted analyses from a large cohort of male health professionals, 50–85 years old and initially free of CVD, revealed that a moderate degree of outwardly expressed anger was significantly associated with a decreased risk of nonfatal MI in the total sample (Eng, Fitzmaurice, Kubzansky, Rimm, & Kawachi, 2003). In men younger than 65 years of age, high and moderate degrees of outwardly expressed anger were protective of total CVD. Length of follow-up was 2 years.

25.2.2 Stroke

Mixed results have also been reported for the relationship of anger expression/control to stroke. In the Kuopio Ischemic Heart Disease Study, this relationship was assessed among 2,074 middle-aged Finnish men who were 53 years of age, on average, at study entry (Everson et al., 1999). Prospective analyses indicated that across an average of 8.3 years of follow-up men in the highest category of outwardly expressed anger with preexisting CHD were nearly seven times as likely to have a stroke (ischemic or hemorrhagic) compared to their counterparts in the lowest category. These effects were independent of potential biologic and sociodemographic confounders. In multivariate-adjusted analyses of male health professionals who were free of CVD at baseline, a high degree of outwardly expressed anger was found to be protective of nonfatal and total stroke over a 2-year follow-up period (Eng et al., 2003).

25.2.3 Atherosclerosis

Consistent with findings on trait anger/chronic hostility, studies support a positive association between anger expression/control and subclinical atherosclerosis. In cross-sectional analyses of middle-aged Korean adults, aged 40–60 years, the anger total subscale of the Spielberger Anger Expression Scale (inclusive of anger in and anger out) was significantly associated with coronary artery calcification (Koh, Choe, & An, 2003). These analyses were adjusted for age, gender, and the presence or absence of CHD risk factors. Over a 2-year period, middle-aged Finnish men with high cynical distrust and high anger control were found to have twice the amount of carotid artery atherosclerosis compared to their low cynical distrust and low anger control counterparts (Julkunen et al., 1994). This effect was independent of age, cigarette smoking, LDL cholesterol, socioeconomic status, baseline carotid artery IMT, season of baseline examination, and days of follow-up. In middle-aged postmenopausal women, aged 42–50 years at study entry, anger in and hostile attitudes predicted carotid IMT over an average of 10 years, independent of the traditional CVD risk factors (Matthews, Owens, Kuller, Sutton-Tyrrell, & Jansen-McWilliams, 1998). In addition, investigators from the Baltimore Longitudinal Study on Aging examined the associations of trait anger and anger expression style to carotid artery IMT and carotid artery stiffness in a sample of men and women who were over the age of 50 (Anderson, Metter, Hougaku, & Najjar, 2006). Cross-sectional analyses from this study revealed that high anger in was positively and independently associated with carotid artery stiffness and carotid artery IMT.

The majority of the studies reviewed indicate that anger expressed outwardly, anger controlled, and anger held in are each positively associated with CVD, independent of the traditional risk factors. One study showed a protective effect of outwardly expressed anger on CHD and stroke among men of high socioeconomic status, although the restricted educational range of the participants may limit the generalizability of these findings. On balance, the data indicate that anger expression of all types is associated with adverse cardiovascular outcomes.

25.3 Acute Anger Episodes

For centuries, emotional distress, such as intense, episodic anger, has been thought to precipitate acute coronary events. Anecdotal evidence of this notion abounds. Perhaps the most famous example is that of Dr. John Hunter (1728–1793), the British surgeon and anatomist, who is quoted as saying, “My life is in the hands of any rascal who chooses to annoy and tease me” (Castiglioni, 1947).

Dr. Hunter frequently experienced anginal symptoms that followed bouts of extreme emotional distress. He died suddenly after being angered in a board meeting at St. George's Hospital. Empirically, anger has been investigated as a trigger of acute MI among 1,623 men and women (61.3 years of age, on average) who were enrolled in the Determinants of Myocardial Onset Study (Mittleman et al., 1995). The investigators used a unique methodology called a case-crossover study design in which participants were asked to recall anger-provoking events that occurred at three different times – the period immediately preceding the MI, the same time period the day before the MI, and the preceding year. The intensity of each episode was rated on a scale from 1 (Calm) to 7 (Enraged, lost control, throwing objects, hurting yourself or others). Results showed that participants who displayed intense anger in the 2 h preceding an MI were at more than twice the risk for an event compared to their experience with anger in the preceding year. A relative risk of 4.0 was reported when intense anger in the 2 h preceding an MI was compared to the same time period the day before the MI.

Use of a case-crossover design such as the one described above provides an exciting opportunity to observe the immediate effects of intense, episodic anger. This design permits an assessment of anger that is proximate to the event. In so doing, it implicates a direct, pathophysiological mechanism and suggests that anger may be a catalyst for hemodynamic and neurohormonal activity resulting in an acute coronary event.

25.4 Putative Mechanisms

Anger/hostility is associated with CVD through a complex set of interrelated factors. Two major hypotheses have been advanced to explain this association (Rozanski, Blumenthal, & Kaplan, 1999). One is behavioral and asserts that highly angry/hostile people are vulnerable by virtue of their propensity for deleterious lifestyles that are known to initiate and accelerate CVD. The corollary is a more adverse CVD risk profile. The second hypothesis implicates a direct, physiological mechanism via the hemodynamic and neurohormonal responses of the sympathetic adrenomedullary system and of the hypothalamic pituitary adrenal (HPA) axis.

Results from several analyses support the behavioral hypothesis. In a recent meta-analysis of 27 studies that assessed the relationship between hostility, as measured by the Cook-Medley Hostility Scale, and CVD risk factors, the authors concluded that hostility was significantly associated with the lipid ratio (total cholesterol/HDL), triglycerides, body weight, insulin resistance, glucose levels, alcohol consumption, and cigarette smoking (Bunde & Suls, 2006). Further, in a long-term follow-up study among college students, higher levels of hostility were associated with increased cigarette smoking and increased consumption of alcoholic beverages 30 years later (Siegler et al., 2003). There are also reports of positive relationships between anger/hostility and other CVD risk factors, such as ambulatory blood pressure levels (Schum, Jorgensen, Verhaeghen, Sauro, & Thibodeau, 2003), hypertension (Yan et al., 2003), and components of the metabolic syndrome (Nelson et al., 2004; Niaura et al., 2000). Further, anger out scores have been positively associated with LDL-cholesterol levels and body weight (Rutledge et al., 2001). Negative associations between anger expression and ambulatory diastolic blood pressure levels (Schum et al., 2003) and between hostility and HDL-cholesterol levels (Chaput et al., 2002) have been reported.

The second hypothesis that has been formulated to explain the anger/hostility–CVD association implicates a direct, physiological mechanism – one that promotes prothrombotic and atherosclerotic changes that play an important role in CVD. At its core, the experience of anger/hostility is a stress response. As a stress response, the physiological consequences of anger and hostility are thought to be mediated by the sympathetic adrenomedullary system and the hypothalamic pituitary adrenocortical (HPA) axis (Chapter 7 by G. Stemmler, this book). Heightened sympathetic activity, most notably

excess circulating catecholamines (epinephrine and norepinephrine), is associated with increased platelet aggregation and with increased blood pressure and heart rate reactivity. Cardiovascular reactivity can cause injury to the heart muscle, endothelial damage, cardiac rhythm disturbances, and disruption of vulnerable plaques. Heightened HPA activity, via the release of adrenocorticotropin hormone, is associated with the secretion of cortisol, which promotes abdominal adiposity, increased insulin resistance, lipid abnormalities, and immune suppression (Black, 2003). Both stress response systems (sympathetic nervous system and the HPA axis) can promote inflammation (e.g., activation of macrophages, the production of cytokines, activation of acute phase proteins, and mast cell activation), which plays an important role in atherogenesis.

The direct, pathophysiological hypothesis has also been supported empirically. Laboratory studies have shown that in stressful situations high-hostile men respond with cardiovascular reactivity, platelet reactivity (Markovitz, Matthews, Kiss, & Smitherman, 1996), and heightened neuroendocrine activity (Suarez, Kuhn, Schanberg, Williams, & Zimmerman, 1998). There is also some indication that high-hostile men and women have more robust and more prolonged blood pressure reactivity in the presence of anger-provoking situations (Fredrickson et al., 2000). Further, daytime cortisol levels among high-hostile men have been shown to be higher compared to their morning waking levels and higher compared to the daytime levels of their low-hostile counterparts (Pope & Smith, 1991).

Several studies indicate that anger/hostility is positively associated with proinflammatory cytokines that have been shown to be related to adverse cardiac events. In one investigation, anger, hostility, severity of depressive symptoms as well as the combined effect of these factors were positively related to plasma IL-6 levels among men not taking multivitamin supplements (Suarez, 2003a). In another, hostility was associated with IL-6 and tumor necrosis factor (TNF- α) among men and women with low depression (Miller, Freedland, Carney, Stetler, & Banks, 2003). As the level of depression increased, the association of hostility with the proinflammatory cytokines decreased. Heterogeneity of association by depression was observed in another analysis; however, in this instance and in contrast to the results of the previous study, hostility was associated with IL-6 levels in men who had high levels of depression (Suarez, 2003). Further, a positive association was observed between hostility and lipopolysaccharide-stimulated (LPS) expression of IL-1 α , IL-8, and IL-1 β among women (Suarez, Lewis, Krishnan, & Young, 2004). In them, the combination of hostility and depression was positively associated with IL-1 β , IL-8, TNF- α , and IL-1 α . Similar results were observed in men, among whom higher hostility was associated with greater levels of LPS-stimulated TNF- α (Suarez, Lewis, & Kuhn, 2002). Additional studies have shown positive associations of hostility with C-reactive protein levels, an acute phase reactant (Graham et al., 2006; Coccaro 2006; Suarez, 2004).

Several reports attest to the significant cardiac and vascular changes engendered by stress and aggression, thereby supporting the biologic plausibility of an anger/hostility-CVD association (Chapter 14 by J.A. Hubbard et al., this book). Experimentally induced psychosocial stress caused endothelial injury in cynomolgus monkeys (Skantze et al., 1998). Experimentally induced aggression in dogs caused an increase in T-wave alternans, indicating electrical instability of the heart (Kovach, Nearing, & Verrier, 2001), and triggered myocardial ischemia in previously stenosed arteries (Verrier, Hagestad, & Lown, 1987). In humans, laboratory-induced mental stress was associated with greater vascular resistance among men and women with low endothelium-dependent arterial dilation (Sherwood, Johnson, Blumenthal, & Hinderliter, 1999). Among men, mental stress was associated with diminished brachial artery flow-mediated dilation at 30 and 90 min after a 5-min speaking task (Ghiadoni et al., 2000). In a study of patients with coronary artery disease, anger recall was associated with vasoconstriction in stenosed coronary arteries (Boltwood, Taylor, Burke, Grogin, & Giacomini, 1993). Finally, in a longitudinal analysis of postmenopausal women, Type

A/anger assessed at baseline was associated with impaired brachial artery dilation an average of 13.6 years later, owing primarily to the influence of Type A behavior (Harris, Matthews, Sutton-Tyrrell, & Kuller, 2003).

There is still lack of clarity regarding the precise mechanism that is responsible for the anger/hostility–CVD relationship. As evident from the discussion above, there is support for both an indirect, behavioral and a direct, pathophysiological mechanism. As for the role of the CVD risk factors, ultimately, the answer may hinge on the nature of the risk profile itself – including not only the number of factors present but also the severity of disease and its clinical trajectory.

25.5 Summary and Conclusions

Studies continue to confirm a positive association between anger/hostility and CVD. Consistent with earlier findings, there are some recent indicators that the anger/hostility–CVD relationship is stronger in younger ages. In the population-based studies, the trend was for an increased incidence of CVD among those with high anger/hostility. However, among the large prospective cohort studies of major CVD events, women and members of racial/ethnic minority groups remain underrepresented, and therefore, gender and racial/ethnic disparities still have not been adequately addressed. This is unfortunate.

Regarding anger expression/control, the majority of studies show that all three expression characteristics are positively associated with CVD. Thus, the question remains as to whether one modality has more adverse CVD consequences than another. In the final analysis, the mere *experience* of the intense physiological arousal associated with anger may be the predominant influence on cardiovascular health; the subsequent mode of expression may be relatively inconsequential. The mechanisms that underlie the anger/hostility–CVD link are complex and have not been fully clarified. There is still the question of whether the association is independent of the risk factors or mediated by them. The evidence from acute effects shows that anger is sufficient to precipitate a coronary event. For some individuals, the operative mechanisms may not be mutually exclusive – that is, CVD may be the final common pathway for a complex array of interrelated behavioral and physiological forces that are induced by anger/hostility. In the setting of risk factor clustering, protracted disease trajectories, and advanced disease states, a behavioral mechanism might be more likely; in low-risk populations, the mechanism might be direct. Finally, while most large, prospective studies have examined the risk factors as possible confounders, very few have reported their modifying effects.

References

- Anderson, D. E., Metter, E. J., Hougaku, H., & Najjar, S. S. (2006). Suppressed anger is associated with increased carotid arterial stiffness in older adults. *American Journal of Hypertension*, 19(11), 1129–1134.
- Appels, A. (1997). Why do imminent victims of a cardiac event feel so tired? *International Journal of Clinical Practice*, 51(7), 447–450.
- Beaglehole, R., & Magnus, P. (2002). The search for new risk factors for coronary heart disease: Occupational therapy for epidemiologists? *International Journal of Epidemiology*, 31(6), 1117–1122.
- Black, P. H. (2003). The inflammatory response is an integral part of the stress response: Implications for atherosclerosis, insulin resistance, type II diabetes and metabolic syndrome X. *Brain, Behavior, and Immunity*, 17(5), 350–364.
- Bleil, M. E., McCaffery, J. M., Muldoon, M. F., Sutton-Tyrrell, K., & Manuck, S. B. (2004). Anger-related personality traits and carotid artery atherosclerosis in untreated hypertensive men. *Psychosomatic Medicine*, 66(5), 633–639.
- Boltwood, M. D., Taylor, C. B., Burke, M. B., Grogan, H., & Giacomini, J. (1993). Anger report predicts coronary artery vasomotor response to mental stress in atherosclerotic segments. *American Journal of Cardiology*, 72(18), 1361–1365.

- Bunde, J., & Suls, J. (2006). A quantitative analysis of the relationship between the Cook-Medley Hostility Scale and traditional coronary artery disease risk factors. *Health Psychology, 25*(4), 493–500.
- Castiglioni, A. (1947). *A history of medicine* (pp. 596–598). New York: Alfred A. Knopf.
- Chang, P. P., Ford, D. E., Meoni, L. A., Wang, N., & Klag, M. J. (2002). Anger in young men and subsequent premature cardiovascular disease: The Precursors Study. *Archives of Internal Medicine, 162*(8), 901–906.
- Chaput, L. A., Adams, S. H., Simon, J. A., Blumenthal, R. S., Vittinghoff, E., Lin, F., et al., for the Heart Estrogen/progestin Replacement Study (HERS) Research Group. (2002). Hostility predicts recurrent events among postmenopausal women with coronary heart disease. *American Journal of Epidemiology, 156*(12), 1092–1099.
- Coccaro, E. F. (2006). Association of C-reactive protein elevation with trait aggression and hostility in personality disordered subjects: A pilot study. *Journal of Psychiatric Research, 40*(5), 460–465.
- Eaker, E. D., Sullivan, L. M., Kelly-Hayes, M., D'Agostino, R. B., & Benjamin, E. J. (2004). Anger and hostility predict the development of atrial fibrillation in men in the Framingham Offspring Study. *Circulation, 109*(10), 1267–1271.
- Eng, P. M., Fitzmaurice, G., Kubzansky, L. D., Rimm, E. B., & Kawachi, I. (2003). Anger expression and risk of stroke and coronary heart disease among male health professionals. *Psychosomatic Medicine, 65*(1), 100–110.
- Everson, S. A., Kaplan, G. A., Goldberg, D. E., Lakka, T. A., Sivenius, J., & Salonen, J. T. (1999). Anger expression and incident stroke. Prospective evidence from the Kuopio Ischemic Heart Disease Study. *Stroke, 30*(8), 523–528.
- Everson, S. A., Kauhanen, J., Kaplan, G. A., Goldberg, D. E., Julkunen, J., Tuomilehto, J., et al. (1997). Hostility and increased risk of mortality and acute myocardial infarction: The mediating role of behavioral risk factors. *American Journal of Epidemiology, 146*(2), 142–152.
- Everson-Rose, S. A., Lewis, T. T., Karavolos, K., Matthews, K. A., Sutton-Tyrrell, K., & Powell, L. H. (2006). Cynical hostility and carotid atherosclerosis in African American and white women: The Study of Women's Health Across the Nation (SWAN) Heart Study. *American Heart Journal, 152*(5), 982.e7–982.e13.
- Fredrickson, B. L., Maynard, K. E., Helms, M. J., Haney, T. L., Siegler, I. C., & Barefoot, J. C. (2000). Hostility predicts magnitude and duration of blood pressure response to anger. *Journal of Behavioral Medicine, 23*(3), 229–243.
- Friedman, M., & Rosenman, R. H. (1959). Association of specific overt behavior pattern with blood and cardiovascular findings: Blood cholesterol level, blood clotting time, incidence of arcus senilis, and clinical coronary artery disease. *JAMA, 2169*(12), 1286–1296.
- Gallagher, J. E. J., Yarnell, J. W. G., Sweetnam, P. M., Elwood, P. C., & Stansfeld, S. A. (1999). Anger and incident heart disease in the caerphilly study. *Psychosomatic Medicine, 61*(4), 446–453.
- Ghiadoni, L., Donald, A. E., Cropley, M., Mullen, M. J., Oakley, G., Taylor, M., et al. (2000). Mental stress induces transient endothelial dysfunction in humans. *Circulation, 102*(20), 2473–2478.
- Goodman, M., Quigley, J., Moran, G., Meilman, H., & Sherman, M. (1996). Hostility predicts restenosis after percutaneous transluminal coronary angioplasty. *Mayo Clinic Proceedings, 71*(8), 729–734.
- Graham, J. E., Robles, T. F., Kiecolt-Glaser, J. K., Malarkey, W. B., Bissell, M. G., & Glaser, R. (2006). Hostility and pain are related to inflammation in older adults. *Brain, Behavior, and Immunity, 20*(4), 389–400.
- Haas, D. C., Chaplin, W. F., Shimbo, D., Pickering, T. G., Burg, M., & Davidson, K. W. (2005). Hostility is an independent predictor of recurrent coronary heart disease events in men but not women: Results from a population based study. *Heart, 91*(12), 1609–1610.
- Harris, K. F., Matthews, K. A., Sutton-Tyrrell, K., & Kuller, L. H. (2003). Associations between psychological traits and endothelial function in postmenopausal women. *Psychosomatic Medicine, 65*(3), 402–409.
- Hearn, M. D., Murray, D. M., & Luepker, R. V. (1989). Hostility, coronary heart disease, and total mortality: A 33-year follow-up study of university students. *Journal of Behavioral Medicine, 12*(2), 105–121.
- Helmer, D. C., Ragland, D. R., & Syme, S. L. (1991). Hostility and coronary artery disease. *American Journal of Epidemiology, 133*(2), 112–122.
- Iribarren, C., Sidney, S., Bild, D. E., Liu, K., Markovitz, J. H., Roseman, J. M., et al. (2000). Association of hostility with coronary artery calcification in young adults: The CARDIA Study. *JAMA, 283*(19), 2546–2551.
- Julkunen, J., Salonen, R., Kaplan, G. A., Chesney, M. A., & Salonen, J. T. (1994). Hostility and the progression of carotid atherosclerosis. *Psychosomatic Medicine, 56*, 519–525.
- Kawachi, I., Sparrow, D., Spiro, A., III, Vokonas, P., & Weiss, S. T. (1996). A prospective study of anger and coronary heart disease. *Circulation, 94*(9), 2090–2095.
- Koh, K. B., Choe, K. O., & An, S. K. (2003). Anger and coronary calcification in individuals with and without risk factors of coronary artery disease. *Yonsei Medical Journal, 44*(5), 793–799.
- Kop, W. J. (1997). Acute and chronic psychological risk factors for coronary syndromes: Moderating effects of coronary artery disease severity. *Journal of Psychosomatic Research, 43*(2), 167–181.

- Kovach, J. A., Nearing, B. D., & Verrier, R. L. (2001). Angerlike behavioral state potentiates myocardial ischemia-induced T-wave alternans in canines. *Journal of the American College of Cardiology*, 37(6), 1719–1725.
- Levenson, J. W., Skerrett, P. J., & Gaziano, J. M. (2002). Reducing the global burden of cardiovascular disease: The role of risk factors. *Preventive Cardiology*, 5(4), 188–199.
- Markovitz, J. H., Matthews, K. A., Kiss, J., & Smitherman, T. C. (1996). Effects of hostility on platelet reactivity to psychological stress in coronary heart disease patients and in healthy controls. *Psychosomatic Medicine*, 58(2), 143–149.
- Matthews, K. A., Glass, D. C., Rosenman, R. H., & Bortner, R. W. (1977). Competitive drive, pattern A, and coronary artery disease: A further analysis of some data from the Western Collaborative Group Study. *Journal of Chronic Diseases*, 30(8), 489–498.
- Matthews, K. A., Gump, B. B., Harris, K. F., Haney, T. L., & Barefoot, J. C. (2004). Hostile behaviors predict cardiovascular mortality among men enrolled in the Multiple Risk Factor Intervention Trial. *Circulation*, 109(1), 66–70.
- Matthews, K. A., Owens, J. F., Kuller, L. H., Sutton-Tyrrell, K., & Jansen-McWilliams, L. (1998). Are hostility and anxiety associated with carotid atherosclerosis in healthy postmenopausal women? *Psychosomatic Medicine*, 60(5), 633–638.
- McCranie, E. W., Watkins, L. O., Brandsma, J. M., & Sisson, B. D. (1986). Hostility, coronary heart disease (CHD) incidence, and total mortality: Lack of association in a 25-year follow-up study of 478 physicians. *Journal of Behavioral Medicine*, 9(2), 119–125.
- Meesters, C. M. G., & Smulders, J. (1994). Hostility and myocardial infarction in men. *Journal of Psychosomatic Research*, 38(7), 727–734.
- Mendes de Leon, C. F., Kop, W. J., de Swart, H. B., Bar, F. W., & Appels, A. P. (1996). Psychosocial characteristics and recurrent events after percutaneous transluminal coronary angioplasty. *American Journal of Cardiology*, 77(4), 252–255.
- Miller, G. E., Freedland, K. E., Carney, R. M., Stetler, C. A., & Banks, W. A. (2003). Cynical hostility, depressive symptoms, and the expression of inflammatory risk markers for coronary artery disease. *Journal of Behavioral Medicine*, 26(6), 501–515.
- Miller, T. Q., Smith, T. W., Turner, C. W., Gujjarro, M. L., & Hallet, A. J. (1996). A meta-analytic review of research on hostility and physical health. *Psychological Bulletin*, 119(2), 322–348.
- Mittleman, M. A., Maclure, M., Sherwood, J. B., Mulry, R. P., Tofler, G. H., Jacobs, S. C., et al., for the Determinants of Myocardial Infarction Onset Study Investigators. (1995). Triggering of acute myocardial infarction onset by episodes of anger. *Circulation*, 92(7), 1720–1725.
- Nelson, T. L., Palmer, R. F., & Pedersen, N. L. (2004). The metabolic syndrome mediates the relationship between cynical hostility and cardiovascular disease. *Experimental Aging Research*, 30(2), 163–177.
- Niaura, R., Banks, S. M., Ward, K. D., Stoney, C. M., Spiro, A., III, Aldwin, C. M., et al. (2000). Hostility and the metabolic syndrome in older males: The Normative Aging Study. *Psychosomatic Medicine*, 62(1), 7–16.
- O'Malley, P. G., Jones, D. L., Feuerstein, I. M., & Taylor, A. J. (2000). Lack of correlation between psychological factors and subclinical coronary artery disease. *The New England Journal of Medicine*, 343(18), 1298–1304.
- Pope, M. K., & Smith, T. W. (1991). Cortisol excretion in high and low cynically hostile men. *Psychosomatic Medicine*, 53(4), 386–392.
- Räikkönen, K., Matthews, K. A., Sutton-Tyrrell, K., & Kuller, L. H. (2004). Trait anger and the metabolic syndrome predict progression of carotid atherosclerosis in healthy middle-aged women. *Psychosomatic Medicine*, 66(6), 903–908.
- Rozanski, A., Blumenthal, J. A., & Kaplan, J. (1999). Impact of psychological factors on the pathogenesis of cardiovascular disease and implications for therapy. *Circulation*, 99(16), 2192–2217.
- Rutledge, T., Reis, S. E., Olson, M., Owens, J., Kelsey, S. F., Pepine, C. J., et al. (2001). Psychosocial variables are associated with atherosclerosis risk factors among women with chest pain: The WISE Study. *Psychosomatic Medicine*, 63(2), 282–288.
- Schum, J. L., Jorgensen, R. S., Verhaeghen, P., Sauro, M., & Thibodeau, R. (2003). Trait anger, anger expression, and ambulatory blood pressure: A meta-analytic review. *Journal of Behavioral Medicine*, 26(5), 395–415.
- Sherwood, A., Johnson, K., Blumenthal, J. A., & Hinderliter, A. L. (1999). Endothelial function and hemodynamic responses during mental stress. *Psychosomatic Medicine*, 61(3), 365–370.
- Siegler, I. C., Costa, P. T., Brummett, B. H., Helms, M. J., Barefoot, J. C., Williams, R. B., et al. (2003). Patterns of change in hostility from college to midlife in the UNC Alumni Heart Study predict high-risk status. *Psychosomatic Medicine*, 65(5), 738–745.

- Skantze, H. B., Kaplan, J., Pettersson, K., Manuck, S., Blomqvist, N., Kyes, R., et al. (1998). Psychosocial stress causes endothelial injury in cynomolgus monkeys via β_1 -adrenoceptor activation. *Atherosclerosis*, 136(1), 153–161.
- Suarez, E. C. (2003). Plasma interleukin-6 is associated with psychological coronary risk factors: Moderation by use of multivitamin supplements. *Brain, Behavior, and Immunity*, 17(4), 296–303.
- Suarez, E. C. (2003). Joint effect of hostility and severity of depressive symptoms on plasma interleukin-6 concentration. *Psychosomatic Medicine*, 65(4), 523–527.
- Suarez, E. C. (2004). C-reactive protein is associated with psychological risk factors of cardiovascular disease in apparently healthy adults. *Psychosomatic Medicine*, 66(5), 684–691.
- Suarez, E. C., Kuhn, C. M., Schanberg, S. M., Williams, R. B., Jr., & Zimmerman, E. A. (1998). Neuroendocrine, cardiovascular, and emotional responses of hostile men: The role of interpersonal challenge. *Psychosomatic Medicine*, 60(1), 78–88.
- Suarez, E. C., Lewis, J. G., Krishnan, R. R., & Young, K. H. (2004). Enhanced expression of cytokines and chemokines by blood monocytes to in vitro lipopolysaccharide stimulation are associated with hostility and severity of depressive symptoms in healthy women. *Psychoneuroendocrinology*, 29(9), 1119–1128.
- Suarez, E. C., Lewis, J. G., & Kuhn, C. (2002). The relation of aggression, hostility, and anger to lipopolysaccharide-stimulated tumor necrosis factor (TNF)-alpha by blood monocytes from normal men. *Brain, Behavior, and Immunity*, 16(6), 675–684.
- Verrier, R. L., Hagestad, E. L., & Lown, B. (1987). Delayed myocardial ischemia induced by anger. *Circulation*, 75(1), 249–254.
- Williams, J. E., Couper, D. J., Din-Dzietham, R., Nieto, F. J., & Folsom, A. R. (2007). Race-gender differences in the association of trait anger to subclinical carotid artery atherosclerosis: The Atherosclerosis Risk in Communities (ARIC) Study. *American Journal of Epidemiology*, 165(11), 1296–1304.
- Williams, J. E., Din-Dzietham, R., & Szklo, M. (2006). Trait anger and arterial stiffness: Results from the Atherosclerosis Risk in Communities (ARIC) Study. *Preventive Cardiology*, 9(1), 14–20.
- Williams, J. E., Nieto, F. J., Sanford, C. P., Couper, D. J., & Tyroler, H. A. (2002). The association between trait anger and incident stroke risk: The Atherosclerosis Risk in Communities (ARIC) Study. *Stroke*, 33(1), 13–20.
- Williams, J. E., Nieto, F. J., Sanford, C. P., & Tyroler, H. A. (2001). Effects of an angry temperament on coronary heart disease risk: The Atherosclerosis Risk In Communities (ARIC) Study. *American Journal of Epidemiology*, 154(3), 230–235.
- Williams, J. E., Paton, C. C., Siegler, I. C., Eigenbrodt, M. L., Nieto, F. J., & Tyroler, H. A. (2000). Anger proneness predicts incident coronary heart disease risk: Prospective analysis from the Atherosclerosis Risk in Communities (ARIC) Study. *Circulation*, 101(17), 2034–2039.
- Yan, L. L., Liu, K., Matthews, K. A., Daviglus, M. L., Ferguson, T. F., & Kiefe, C. I. (2003). Psychosocial factors and risk of hypertension: The Coronary Artery Risk Development in Young Adults (CARDIA) Study. *JAMA*, 290(16), 2138–2148.

Chapter 26

The Anger of Pain Sufferers: Attributions to Agents and Appraisals of Wrongdoings

Ephrem Fernandez and Ajay Wasan

Abstract In this chapter, anger is viewed as a major element in the affective component of pain. How this anger comes about is a matter of some debate. While it has been speculated that pain and anger are hardwired, our critical analysis of the evidence raises several definitional problems and alternative interpretations. Even sudden noxious stimuli are subject to information processing in a variety of ways including “personification.” In the more common and pervasive case of chronic pain, it is evident that pain does not occur in a vacuum but exerts far-reaching effects on occupational, recreational, social, interpersonal, and self-care functions. These become the material for appraisals of wrongdoing and attributions of anger. The many targets of this anger are surveyed, including health-care providers, mental health professionals, insurance carriers, and even significant others. Apart from such anger as an appraised consequence of pain, other possible interactions between the two are outlined. As explained, anger may also function as a predisposing factor, a precipitant, an exacerbating factor, and a perpetuating factor in pain. This framework accounts for the many complex and diverse interactions between pain and anger and also directs us to different pathways to the alleviation of pain and anger in populations with medical ailments.

26.1 Introduction

At the intersection of affect science and health research is a body of evidence that negative affect, in particular anger, prevails in medical populations such as those in pain. In an attempt to explore and explain the anger of pain sufferers, we begin by clarifying the concept of pain and presenting some of its epidemiological statistics. This is followed by a definition of anger and related concepts. Against this conceptual backdrop, it becomes possible to shed light on the origins of anger in pain sufferers in addition to outlining the different ways in which anger and pain can interact.

Within scientific discourse, the word pain is used to denote something somatic, other usages usually being metaphors of that word pain. Put simply, pain is that which is identified with reference to some anatomical location in or on the body. It is not pain in the loose sense of the word as

E. Fernandez (✉)

Department of Psychology, University of Texas at San Antonio, San Antonio, TX 78249, USA
e-mail: ephrem.fernandez@utsa.edu

when people complain of the “pain” of failure, the “pain” of loss or the “pain” of yearning for a distant loved one. No doubt these are legitimate sources of suffering but to call them pain would be somewhat of a figure of speech, quite admissible in literary writing. For our present purposes, it pays to stick to the current convention within the life sciences where the term pain is reserved for aversive physical sensations that are associated with tissue damage (International Association for the Study of Pain, 1986). This not only brings some focus to the discussion but it also avoids a confounding of the mechanisms for pain-related suffering with the mechanisms of general distress or negative affect unrelated to pain.

Pain is a global problem, with one in five persons suffering moderate to severe levels of chronic pain, many of them living in lower income countries where chronic diseases abound (World Health Organization, 2004). Chronic pain can be defined as pain lasting at least for 3 months and experienced daily though not necessarily all day long (Magni, Marchetti, Moreschi, Merskey, & Luchini, 1993). Within the USA, about 25 million people suffer acute pain from surgery or accidents and a total of 50 million live with chronic pain due to disease, disorder, or accident (American Academy of Pain Management, 2006). Pain is easily the chief complaint in medical emergency departments (Cordell et al., 2002) and is now deemed to be the most common reason why people seek medical care. The ubiquity and magnitude of pain have prompted professional organizations (e.g., the American Pain Society, the Joint Commission on Accreditation of Healthcare Organizations) to recommend that pain be assessed as “the fifth vital sign” along with pulse, blood pressure, respiration, and body temperature.

It is the functional impairment from pain (in addition to the pain itself) that attracts the interest and effort of psychologists and many allied health-care professionals. Pain does not occur in a vacuum but exerts far-reaching effects on occupational, recreational, social, interpersonal, and self-care functions. In fact, the economic burden of chronic pain has been estimated at \$90 billion in the USA alone (National Chronic Pain Outreach Association, 2006). Of course, this barely accounts for the immeasurable suffering brought on by painful conditions. Suffering is manifold and can take the form of discrete emotions such as fear, sadness, or anger which may be drawn out into clinical levels of anxiety, depression, and maladaptive anger (Fernandez, 2002; Fernandez, Clark, & Rudick-Davis, 1999; Wasan, Fernandez, Jamison, & Bhattacharya, 2007; Wasan, Gudarz, & Jamison, 2005). The last of these, anger, is often a special element in the suffering of pain patients and it falls within the focus of this chapter.

Despite a voluminous literature on anxiety and depression as related to pain, research on anger in relation to pain has been recent in history and slow to accumulate. This is probably due in no small degree to the underrepresentation of anger in psychiatric nosologies and its virtual absence as a diagnostic category in the American Psychiatric Association’s Diagnostic and Statistical Manual of Mental Disorders. As a first step toward rectifying this oversight and recognizing the place of anger in pain, we offer a definition of anger. In doing so, we not only explicate the components of anger but also delineate the boundaries between anger and other related concepts.

26.2 Phenomenon of Anger

Anger is best situated among the various feeling-related phenomena, all of which have quality, quantity, and form (Fernandez, 2008; Fernandez & Kerns, 2008). The qualitative aspect of emotion is sometimes referred to as valence. Qualitatively, anger is always an unpleasant feeling because disapproval is embedded in it. In that sense, it has something in common with other types of unpleasant affect like sadness and fear, though as will be shown later, there are other distinguishing features of

each. The quantitative dimension of emotion pertains to intensity. Quantitatively, anger varies on a continuum from low levels of magnitude that may be called annoyance to high levels called rage or fury (Chapter 22 by Potegal, this book). In addition, anger (like fear and sadness) can assume the form of an emotion, a mood, or a temperament, depending on whether its pattern of occurrence is phasic, tonic, or cyclic, respectively. At a psychiatric level, any of these forms can reach the proportions of a clinical disorder.

Structurally, anger, like all emotions, can be deconstructed into a unique pair of cognitive and motivational components. Based on the contributions of several scholars of affect science (Frijda, 1986; Lazarus, 1991, 2000; Ortony, Clore, & Collins, 1988; Scherer, Schorr, & Johnstone, 2001; Smith & Ellsworth, 1985, Chapter 15 by Wranik and Scherer, this book), it can be asserted that the cognitive appraisal inherent in anger is that one has been wronged or offended while the motivation or action tendency is to redress or correct the wrong. This is congruent with commonsense views of anger as revealed through a semantic analysis called *psychologic* (Smedslund, 1988). In his treatise on anger, Smedslund has found lay consensus that anger is “a feeling involving a *belief* that a person one cares for has, intentionally or through neglect, been treated without respect, and a *want* to have that respect re-established” (Smedslund, 1992, p. 30). As noted by Fernandez (2005), the wrongdoing is not always open to objective verification. Rather, it is interpreted or appraised. Justifiably or not, one is aggrieved or takes offense at what appears to be aggression, exploitation, abandonment, deceit, insult, or some other violation of propriety. This sets in motion the urge to counteract the perceived wrongdoing with responses that may range from overt retaliation to passive resistance. It is not the counteractive measure but the tendency to such action that defines anger. In other words, we are adopting a cognitive-motivational rather than a cognitive-behavioral conceptualization of anger.

26.2.1 Anger Versus Aggression

The above definition lays the groundwork for demarcating the boundary between anger as a cognitive-motivational process and aggression as a behavioral outcome. Aggression is behavior that is intended to injure or damage (Worchel, Cooper, Goethals, & Olson, 2000) in terms of bodily harm, psychological hurt, and/or material damage. Aggressive acts may culminate out of action tendencies linked to anger. But, it must not be overlooked that the possible motives for aggression are multifarious, e.g., thrill (as in pyromania), lust (as may be the case in rape), or sheer greed (as in armed robbery).

Just as aggression can occur without anger, the converse, anger without aggression, is well within the scope of possibilities. Many people experience anger without acting on it or even exhibiting it.

26.3 The Anger of Pain: Where Is the Provocation or Wrongdoing?

Based on an appraisal theory of emotion (Scherer et al., 2001), where there is anger, there is perceived provocation or wrongdoing. The obverse also holds true: there is no anger without perceived wrongdoing. Any wrongdoing requires a wrongdoer (but see Chapter 16 by Berkowitz, this book). The angered individual may take offense at the actions of another person, group, self, or thing. Before we discuss those allegedly responsible for the anger of pain patients, let us briefly consider the challenge, sometimes posed, that anger can occur without perceived wrongdoing and human agency. This possibility has bearing on our basic definition of anger as well as on our attempts to explain the anger of pain patients.

26.3.1 Pain Personified

Can one not get angry at the sudden pouring rain, the unrelenting heat, or other vagaries of the weather? Don't people protest angrily at malfunctioning computers, vending machines, and other inanimate objects? Haven't we observed people cursing in anger upon stubbing their toe against the door, or being struck on the head by bird droppings, or stepping into mud – clearly chance occurrences that cannot be attributed to any wrongdoing as such?

In literary works, the allegory has long been used to depict anger and other emotional exchanges between humans and the elements or other non-living things. More than a 100 years ago, G. Stanley Hall (first president of the American Psychological Association) published an empirical paper on anger in which he devoted a subsection to "Anger at inanimate and insentient objects" (p. 565). In Chapter 16 by Berkowitz, this volume while recognizing the atypicality of such anger, has pointed out that conventional appraisal theories should but fail to account for these phenomena.

It is my view (EF) that appraisal theory is applicable even to the atypical situations I have described above. In such situations, the angry reactions are quick and brief and during that time, it is proposed that there is a subconscious personification of the stimulus. Almost reflexively, we are ready to blame and find a culprit and so we do. For a moment or two, it is as if the computer comes to life, it has volition, and it is striving to frustrate or provoke us. As Smedslund says, people tend to regress under stress, but contrary to his presumption, it is not uncommon for people to act upon such anger toward non-persons. This is even more likely when the machine possesses humanoid attributes. For example, modern-day interactive voice response (IVR) systems which are used to give prompts and feedback to customers are often met with a certain phone rage: some customers respond with expletives and angry statements despite the knowledge that the IVR prompts and commands are programmed rather than spontaneous or voluntary. The more robots resemble humans in form (e.g., limbs, head) and function (e.g., locomotion, verbalization) the easier it is for a human being to lapse into a personification of these machines during which anger as well as other emotions are expressed. We propose that this process is fundamentally appraisal-driven but because it is ephemeral and not readily recalled the cognition is left unarticulated.

Now to an extension of this reasoning to the anger that is experienced and expressed by people in pain. By virtue of its intrinsic aversiveness and frustration potential, pain can also trigger impulsively angry responses. In a fleeting moment of pain, the sufferer may curse and swear as if the pain itself is the enemy and the offending entity. This is especially the case in acute pain episodes. As explained earlier, we conjecture that the anger originates from a momentary subconscious personification of the noxious stimulus. However, over time and especially in the case of chronic pain, automatic thoughts may give way to voluntary interpretations. The sufferer may then be in a better position to reason and come to the realization that the pain is at most a signal or symbol of much else that has gone wrong.

26.3.2 Anger Deflected

Sometimes, anger may be redirected from the wrongdoer to a relatively innocent target. This may be termed deflection or what in traditional Freudian nomenclature was known as displacement. Instead of delving into ego defense mechanisms, it may suffice to say that deflection is simply adaptive when the prime target of anger is unassailable or inaccessible. As a result, the anger that lingers is left with no outlet but to be expressed at a target of convenience. This target may be someone remotely associated with the culprit or even totally innocent.

An example of anger deflection (as opposed to reflection) is taking out one's anger against a child when one has actually been offended by one's spouse. In the context of pain, there are various targets

of convenience that take the place of the original targets of blame. For example, a significant other may become a target of anger that is deflected away from the health-care provider. Alternatively, a physician may become the target of anger that was really meant for the employer. To reflect the anger back at the “offending” employer could incur grievous repercussions and so the victim may find it safer to deflect the anger toward a member of the helping profession.

Unless privy to the origin of such anger, an observer may be left with the misimpression that there is no wrongdoer or wrongdoing and that the anger is without its characteristic appraisals. We argue that the anger has merely been deflected from the culpable to the convenient. Inexplicable and unpredictable as it may seem, the anger may spring from appraisals and even continue to be fueled by appraisals. Over time and with new experiences, additional appraisals may come into being and the patient may take aim at one or more in an array of targets.

26.4 Targets of Anger

On surveying the cognitive topography of anger in pain patients, Fernandez and Turk (1995) identified 10 principal targets of the angry complaints of pain patients. These are the persons responsible for the injury/illness, the medical health-care provider, the mental health professional, the legal system, insurance carrier or third-party payer, the employer, significant other, God, self, and the whole world. As will be discussed next, these are not random targets but ones that emerge out of a myriad of complex interpersonal exchanges in which certain actions are appraised as wrongdoings.

26.4.1 The Arch Perpetrator

An obvious target of any anger is the one who is supposed to have committed the wrongdoing, and in the context of pain, that target is the prime person deemed responsible for the pain-causing injury/disease. It is conceivable that this responsibility may fall upon oneself, though attribution theory tells us that blame for a failure or setback is more likely to be externalized than internalized (Heider, 1958; Shaver, 1970; Harvey & Weary, 1984; Weiner, 1986). Findings from pain research are generally consistent with this theory in that others are often blamed for one's own pain (DeGood & Kiernan, 1996; McParland, Whyte, & Murphy-Black, 2005). Such blame may well serve as a means to cope with adversity, although there are conflicting findings on this issue. In some accident victims, self-blame has been associated with good coping (e.g., Bulman & Wortman, 1977; Hart, Bogner, Whyte, & Polansky, 2003) whereas in others it has been compounded with PTSD (McLean, Clauw, Abelson, & Liberzon, 2005). In cancer patients, blaming oneself turns out to be a predictor of later distress (Glider, & Compas, 1999). The benefits or detriments of blame may be further dependent on (i) whether it is characterological blame or behavioral blame that is being assigned, and (ii) the degree of discrepancy between the perception versus the reality of where the fault resides.

26.4.2 Health-Care Providers

In a survey of pain patients, Okifuji, Turk, and Curran (1999) found that about 60% of them reported anger toward health-care providers. The typical reasons for this anger are diagnostic ambiguity and treatment failure. Chronic pain being etiologically elusive, may be the subject of many disagreements about its underlying mechanism. As a rule, 3-month pass before persistent pain is diagnosable as

chronic and even at that point in time the “pathogen” may remain unknown. The search for structural abnormalities may be futile and even when identified they may not be proportionate to the pain as in the classic study of back pain by Jensen et al. (1994) and a more recent study of spinal cord injury patients (Defrin, Ohry, Blumen, & Urca, 2001). Diagnoses themselves may be provisional, undergoing revision over time, and not necessarily gaining consensus across clinicians. When finally announced to the patient, the diagnosis may be little more than a label with minimal explanatory value. The patient is therefore left perplexed and vexed, hardly convinced by assurances that the hurt she/he experiences signals no harm or that the ever present pain has really outlived its function. In the case of myofascial pain, patients have been known to adopt distorted beliefs about their pain and to express dissatisfaction with their physicians (Roth, Horowitz, & Bachman, 1998). This is quite plausibly an offshoot of the notoriously poor patient–doctor communication (Wasan, Wootten, & Jamison, 2005).

The problem of diagnostic ambiguities is addressed in Petrie et al.’s (2005) survey of 42 female and 35 male chronic pain patients in an Auckland, New Zealand, pain clinic. The average pain chronicity was 6 years, the average pain intensity was about 8 on a 10-point scale, the back was the single most common site of pain but about one-third of patients had multiple pain sites. About 25% of patients expected some explanation or improved understanding of their pain problem, making that the singlemost common expectation on the first visit. Unfortunately, the number actually receiving that explanation is unknown but another study of pain patients in the same country revealed a low level of satisfaction attributable in part to poor doctor–patient communication about the diagnosis. (Grace, 1995).

Another attribution of wrongdoing is leveled against the health-care provider when treatment fails to remedy the painful condition. Some statistics convey a positive picture. For example, McCracken, Evon, and Karapas (2002) reported that when surveyed for 6 months following their visit to an anesthesia pain clinic, about 90% of 62 patients were either satisfied or very satisfied with their treatment, about 10% were “somewhat satisfied,” and none were dissatisfied, 70% of the variance in satisfaction being predicted by patients’ perception of how well they had been evaluated, how well the clinic procedures were explained, and their degree of functional improvement. However, these data were based on “completers”; more than two-thirds of those sampled at baseline did not return the questionnaires at follow-up; could this be due to dissatisfaction? Furthermore, the participants in this study provided details of their personal background and the researchers conducted detailed chart reviews of patients’ previous clinic visits, procedures performed, and medications prescribed. In the absence of anonymity, the veridicality of patients’, reports of satisfaction is questionable.

The Petrie et al.’s (2005) survey found that for about 45% of patients, the most satisfying outcome expected of pain clinic consultation was either a cure or some control of their pain. Yet, surgical interventions, if driven by a strictly neurological model, rarely abolish pain and the hopes of pain relief often dwindle with successive operations. From the clinician’s viewpoint, this outcome represents not a failure of treatment but a sign of the intractability of pain: “If treatment fails, your pain is chronic; if your pain is chronic, then treatment fails.” This circularity of reasoning must perplex if not perturb many patients who are already enduring the day-to-day frustrations of unremitting pain. What may be particularly objectionable is the built-in justification for treatment failure – that the problem is in the nature of the pain not the treatment. By extension, it is not the clinician but the patient who has failed. Many patients angrily reject such an outcome. As Petrie et al. (2005) have reported, if pain relief is the common expectation of pain patients, then the most disappointing outcome for a majority of pain patients is no pain relief or being told that nothing can be done. Ironically, more expensive medical treatments for pain are not more helpful to patients than less expensive treatments (Chapman, Jamison, Sanders, Lyman, & Lynch, 2000) and this is likely to be a source of added anger to the pain patient.

26.4.3 Mental Health Professionals

The subsequent entry of the mental health professional into the pain arena is hardly auspicious in the eyes of the chronic pain patient. The insinuation, according to the patient, is that the pain is “in my mind” and that is hard to accept. As eloquently stated by Roy, “Patients regard denial of a physical cause for their pain as demeaning and invocation of psychological explanation as rejection, or at least minimization, of their pain and suffering, or at worst simply questioning their veracity” (2002, p. 11).

This attribution may be further validated if antidepressant or anxiolytic medications are prescribed presumably for premorbid affective disturbance. When prescribing such medications for affective sequelae of pain, the psychiatrist may be regarded as treating symptoms rather than the cause. The psychologist introducing cognitive techniques such as imagery and self-statements may also be viewed unfavorably. Once again, the medium of intervention is the mind whereas the complaint is about the body. Images of pleasant scenery, recital of statements of self-control, and attention–diversion tactics offer many options (Fernandez, 1986) but all too often only enable some coping with a harsh and undeniable reality. Such strategies can be oversold to the point of trivializing the patient’s pain. The alternative of behavior modification has the potential to rehabilitate the patient to a more adaptive level of functioning. However, some of the reinforcement contingencies used to this end introduce their own unique problems. For example, ignoring pain behaviors and reducing solicitude may be misunderstood as callousness. Other behavioral techniques such as setting exercise quotas and tapering medication though evidently efficacious in the long run often elicit resistance and resentment as they increase pain in the short run.

26.4.4 Insurance Carriers, Legal Systems, and Employers

The frustration and anger of the pain sufferer is likely to be augmented several fold if the pain is due to injury on the job, in which case s/he may get embroiled in conflict with the employer, the legal system, and the insurance carrier or third-party payer. In the earlier mentioned survey by Okifuji et al. (1999), attorneys were the target of anger for about 20% of pain patients, insurance companies for about 30% of pain patients, and employers for about 26% of pain patients. These dealings tend to be adversarial, especially when the stakes are high and there is an inordinate burden of proof on the patient to demonstrate impairment and responsibility. Some of these conflictual interactions have been exposed in the USA where “managed care” has made cost containment a priority (Gatchel, 2005; Gatchel & Okifuji, 2006; Robbins et al., 2003). Gatchel and colleagues point out that insurance adjusters, though relatively unschooled or uninterested in the complexities of pain management, have tremendous sway in the authorization and reimbursement of pain care services. Some essential services such as physical therapy are even being “carved out” only to compromise the effectiveness of evidence-based multidisciplinary treatment of pain. In this so-called medical marketplace, the clinician’s authority has been undermined and the patients themselves are often left frustrated and neglected. In the event of termination of a job or mandatory retraining for a new job, life adjustments are likely to bring on stress and financial hardship. By the time there is a settlement on all these fronts, the patient may be consumed or worn out by anger (Roy, 2001). The picture is even more disquieting in third world countries where injury, disability, and pain often lead to loss of employment, little or no monetary compensation, and the collapse of one’s livelihood altogether.

A poignant illustration of anger associated with chronic pain can be seen in a case study provided by Roy (2002). A Canadian woman by the name of Mrs. Abrams, was injured in an automobile accident, after which she developed chronic pain and was unable to keep her job:-

When Mrs. Abrams resigned her position, she lost. . . a vital element of her sense of self. She lost her place. . . as a valued member of a helping profession. Above all, she lost a simple, yet a core, component of her identity. Redefinition of the self was called for, but the answer was far from acceptable. The answer in her case was that of a chronic patient. This radical change in identity extracts an enormous psychological cost. Some patients may experience relief, but not Mrs. Abrams, who took exceptional pride in her profession. She felt humiliated, unfairly treated by the world, sad, and even grief-struck and very angry. (Roy, 2002, p. 4).

Mrs. Abrams' distress was compounded by her insurance carrier's refusal to accept liability for her chronic pain and disability. The company contended that these health problems were unrelated to the accident, even though she had been in relatively good health prior to it and she was convinced that her pain and related problems began afterwards. She reached a financial settlement with the insurance company only after a legal battle lasting several years. Roy notes "It is not an exaggeration to state that much of the dispute between chronic pain patients and their insurance companies, workers compensation boards, and other financial institutions is analogous to Mrs. Abrams' case." (2002, p. 7). Understandably, insurance companies are the object of ire for many chronic pain patients (Fernandez, Salinas, Swift, Iglesias, & Towery, 1995; Fernandez, 1996; Okifuji et al., 1999).

26.4.5 Et Tu Significant Other?

In the path of the pain patient's anger, it is quite surprising to spot many a significant other. These individuals become a target of anger in about 39% of pain patients (Okifuji et al., 1999). Early in the onset of pain, the patient is likely to be the recipient of assistance and nurturance from family and friends. However, this is not always sustainable. As their time and resources are diminished or drained by the ongoing needs of the patient against the competing responsibilities of life, significant others may become less beneficent. Some of them may even turn punitive in which case the emotional aversiveness of the pain experience is intensified McCracken, (2005). The corresponding decline in socio-familial support clashes with the patient's expectations thus producing resentment and conflict. To "add insult to injury," significant others are often advised by health-care staff to withhold or reduce support so that it does not become solicitude. However well-intentioned this plan may be, it is often misconstrued by the patient who may feel entitled to unconditional support from his/her significant others.

Roy (2006, p. 1–2) has reported a case study of a pain patient whose anger seems to have started with his immediate family. It was later exacerbated by the mounting responsibilities from members of his "blended" family in which two previously married individuals enter into a relationship, thus bringing any children of theirs into one reconstituted family:

Mr. Alfred, in his late fifties, . . . , presented at the pain clinic with multiple pain problems including serious osteoarthritis of the knees and shoulder pain of unknown origin. He was angry and hostile during his first visit to the pain clinic, and was indeed very hard to engage in any rational conversation. Over time, however, he cooled off considerably and the reasons for his anger emerged. . . Mr. Alfred was divorced and had single-handedly raised a son and a daughter. He was an authoritarian and demanding father, but. . . very caring and concerned. Both his children were grown up and leading reasonably good lives. He had gotten remarried to a divorced woman who had a daughter in her early twenties, who was very dependent on her mother. In addition, the patient agreed to have his new mother-in-law move in with them. In a rather short. . . time, his life was turned upside down. Not only did he have to learn to make a new life with a new partner, but also he had to adapt to having an elderly dependent person living with them and to the unending demands of his stepdaughter.

. . . Having taken on all his new responsibilities, the patient was not so sure that he had made the right decision. He was filled with rage, but did not have an outlet for it.

Mr. Alfred, already irked by his pain, was enraged by the unanticipated responsibilities of his reconstituted family (an increasingly common phenomenon in Western society). Perhaps the pain lowered the threshold for anger elicited by family members.

26.4.6 *The Whole World and God*

The “fall from grace” of the patient’s social network is only one step away from the patient’s disaffection with the whole world. If kin and kindred spirits let us down, then what is to be expected of the rest? This is when the patient’s anger and alienation may generalize to much or all of society. Moreover, other people who are spared the scourge of pain may be viewed as more fortunate and undeserving than oneself, thus becoming the object of resentment. Also in the mix of anger targets may be Fate or God or a supernatural equivalent. Inasmuch as the patient views himself/herself as having been dealt a bad hand in life, s/he may feel singled out – “why me?” To the extent that the patient regards this as beyond human control, anger is directed at these suprahuman entities, supernatural processes, or abstractions as the case may be.

26.5 The Anger of Pain: Non-cognitive Mechanisms?

Our preceding account provides a picture that the anger of pain patients does not occur in a vacuum; in fact, it is intimately linked to the patients’ social world. Patients appraise the actions of others in ways that lead to anger. Specific attributions of wrongdoing are directed at particular targets ranging from health-care providers through insurance companies to significant others and even God. In recognizing the pivotal role of cognitions patients have about their world, our analysis merges with other examples from theories of social cognition.

However, there are contrary views. It is outside the scope of this chapter to review these but a few studies bear mentioning. Parkinson (1999) has criticized earlier research on self-reported anger on the grounds that demand characteristics may have led participants to provide “rational” explanations for their anger. To test this hypothesis, one group of his subjects reported on anger with good reason (reasonable anger), another reported on anger without good reason (unreasonable anger), and a third group reported on “nonemotional blame” or blame without anger. Aside from the demand characteristics here too, significantly lower anger intensity ratings were found for the nonemotional blame condition than in either of the other two conditions. However, even though 15 out of 47 participants attributed their anger to nonappraisal factors (e.g., rhetorical intent), “other accountability” or blame was rated as the most influential cause of anger. Kuppens, Van Mechelen, Smits, and De Boeck (2003) have reported that perceived arrogance and unfairness were among a handful of appraisals associated with anger but none of these was by itself necessary or sufficient for anger. We agree but add that these appraisals are simply variants of a whole genre of perceived wrongdoings. No particular appraisal will be present in all anger but it is very likely that *at least one* of the appraisals within that genre will be present in any instance of anger.

Recently, Parkinson (2007) re-interpreted the findings of Siemer and Reisenzein (2007) as undermining appraisal theories of emotions. He bases this on the reaction time data from the latter study which indicate that participants made inferences of emotion more quickly than they did appraisal judgments. This may well be because emotions are “wholes,” and by the rules of Gestalt perception, they are more readily perceived than the appraisal details which are parts of the whole. Parkinson

goes on to propose a rather narrow definition of appraisals “If every kind of emotion-relevant situational information necessarily counts as appraisal information, then Seimer and Reisenzein need not have conducted such complicated studies to demonstrate that appraisal information mediates emotion inference. For me, appraisal information constitutes only a subcategory of relational information, and relational information of other kinds can also mediate emotion inference from situation descriptions” (2007, p. 23). In this idiosyncratic view, some bits of information enter into judgments of emotion yet they may not be dressed up as appraisals.

The special case of pain has been considered as a testbed for the alternative position that anger can arise without cognitive mediation. In Chapter 16 by Berkowitz, this volume relates several examples in which the anger of being in pain seems to spring from a source other than the cognitive. Part of the mechanism may reside in cerebral hardwiring (Berkowitz, 1993; Berkowitz & Harmon-Jones, 2004). This may seem like a reasonable explanation especially in the case of acute pain as caused by electric shock, fire, and sudden cuts. Under such circumstances, the withdrawal reflexes in people are so quick as to minimize cognitive mediation. Yet, as we pointed out earlier, there is also the possibility of momentary subconscious personification of the stimulus which is itself a type of cognitive appraisal process. This argument echoes the famous Lazarus–Zajonc debates of the 1970s on the primacy of cognition in emotion. Much of that debate hinged upon definition of what constitutes cognition. If cognition includes any kind of information processing, then the mere attention and recognition entailed in those rapid withdrawal reflexes certainly qualify as cognition. Moreover, at a rudimentary level, the organism must at least be engaging in some appraisal of the valence of the stimulus be it pleasant or unpleasant.

The assumption of cognitively unmediated emotion may seem tenable when we observe infants. When anger first emerges at 4- to 6-month old in human infants, it is typically a result of frustration and lack of instant gratification rather than because of any elaborate meaning attached to the stimulus (Fernandez, 2003). For example, infants show anger when their arms and hands are pinned down as if to immobilize them (Lewis, 2000, this book). Yet, cognitive mediation cannot be ruled out from such scenarios for it is strictly unknowable if the infant perceives some kind of wrongdoing. Even if the anger stems from being frustrated, that in itself must entail a basic perception of conflict or discordance between the infant’s goal and obstacles to the attainment of that goal. As Lewis remarks, the infant must utilize some “means-ends” knowledge at this stage of cognitive development.

With the greater availability of self-report in children and adults, it may still be tempting to negate the existence of appraisals whenever they are not verbalized. This applies to any emotion or affective type. Individuals may say they’re depressed without saying why. Does this mean that their feelings are without appraisals? According to appraisal theory, all feelings entail appraisals (e.g., Roseman, 2004; Smith & Kirby, 2004). As has been explained earlier, the failure to articulate may be due to a variety of reasons such as complexity of the appraisal, unawareness of the appraisal, difficulty of recall, or just plain choice. Additionally, appraisals may occur in such rapid succession and nonlinear fashion that identifying what thoughts precisely make us feel a particular way can be an elusive process. Put simply, an absence of communication does not mean an absence of cognition.

Having said that, we do not disagree with Berkowitz’s and others’ view that feedback from the facial musculature and other parts of the body can intensify emotions such as anger. It would be a mistake to stretch this to a view that anger originates from such bodily events. Berkowitz’s associationistic conception is also worth emphasizing. That cognitive, skeletal–muscular, visceral, and experiential components of emotion are interconnected associatively so that activation of one component can spread to other components, is supported by empirical research. However, we must resist the urge to equate these processes in potency or to assert that there is a specificity with which motoric and autonomic responses activate particular emotions.

26.6 Other Interactions Between Anger and Pain

We now return to the central concern in this treatise: the relationship between anger and pain. There is ample evidence to indicate that the anger of pain sufferers is cognitively mediated. This is especially the case in chronic pain where the anger emerges from a myriad of interpersonal interactions between the patient and others. The anger does not occur in a vacuum or as a hardwired reflex. Rather, its source is in the perceived wrongdoings that pain sufferers attribute to those responsible for the injury/disease, the health-care providers including mental health professionals, the insurance carriers, legal system, and employers. Sometimes, the anger may be “close to home” as when significant others are targeted. Additionally, the anger may have an existential quality in which God and the whole world are blamed or resented. It is possible that anger is directed toward oneself. Finally, through a relatively subconscious process, the pain itself may be personified into an agent with volition. This is transient and with time it usually gives way to more reasoned appraisals.

Hitherto, we have called attention primarily to anger as a consequence of pain. We next outline how anger (or other types of affect) can predispose to, precipitate, exacerbate, and perpetuate pain (Fernandez, 1998, 2002). Alternatively, it may be a mere co-occurrence or correlate of pain.

26.6.1 Anger as Co-occurrent

Perhaps the simplest way of framing a relationship between anger and pain is to say that the two co-occur. Two variables may co-occur at a point in time (episode comorbidity) or else at any time over the life span (lifetime comorbidity). The extent of overlap may be expressed as a percentage or proportion or odds ratios. Comorbidity in the case of pain and anger has been little investigated partly because the only anger-specific diagnosis in DSM is intermittent explosive disorder. This refers to angry/aggressive outbursts that are disproportionate to provoking stimuli. Although rare in the general community, this anger disorder has been diagnosed in about 10% of pain patients (Fishbain, Goldberg, Meagher, & Steele, 1986). In collaboration with the World Health Organization, Kristjansdottir (1997) found that anger was the most common emotional disturbance in 2,400 Icelandic schoolchildren with weekly pain, affecting between 76.5 and 78.3% of the sample. Anger prevalence increased to as much as 85.7% for those with three pains per week. In adult chronic pain patients, anger was reportedly experienced 70% of the time, and two-thirds of this anger was attributable to pain rather than extraneous factors (Fernandez et al., 1999).

26.6.2 Anger as Correlate

Comorbidity data provide a static picture of overlapping variables. A more informative index is the correlation coefficient which indicates the extent to which variables change together. A single study by Gelkopf (1997) found a correlation of 0.60 between anger-in and cold pressor pain sensitivity. Further studies will be required to specify confidence intervals around this statistic along the lines of Dobson's (1985) (now familiar) average correlation of 0.65 between anxiety and depression across studies.

26.6.3 Anger as Predisposing Factor

The correlation coefficient indexes covariation but lacks any sense of directionality of influence. In contrast, the clear temporal succession of events when anger predisposes an individual to pain

implies a causal relationship. More precisely, anger starts as a distal cause that cumulatively results in pain. Such reasoning is common in psychosomatic medicine where various personality factors have been hypothesized to contribute to symptoms of diabetes, cardiovascular disease, and even cancer. The notion of a pain-prone personality was advanced by Engel (1959) and later backed by others like Blumer and Heilbronn (1984) though because it was formulated in traditional psychodynamic theory of infantile needs, it has far less currency today. Nevertheless, recent research has raised the possibility that trait anger may play a role in the genesis of pain (Burns, Higdon, Mullen, Lansky, & Mei Wei, 1999). Connant (1998) has used structural equation modeling to show a statistically significant relationship between trait anger and perceived pain in spinal cord injured patients.

26.6.4 Anger as Precipitant

Just as state anxiety has long been recognized as a sudden trigger of pain, so is state anger likely to precipitate pain. Marcussen and Wolff (1949) found that migraine attacks could be brought on by placing migraine patients in anger-provoking circumstances. Similar findings have been reported in a recent study of both migraine and tension headache (Martin & Teoh, 1999). More recently, Burns, Kubilus, and Bruehl (2003) randomly assigned students to different emotion inductions, one of which was for anger. This was followed by a cold pressor pain test in which temporal measures of threshold and tolerance were obtained along with verbal descriptors on the MPQ. Anger-out (Chapter 23 by Spielberger and Reheiser, this book) had a significant effect on pain sensitivity (but not pain tolerance or MPQ scores) and this effect was paralleled by decreases in systolic blood pressure. While the relative contributions of anger-in versus anger-out to pain intensity are still under investigation, there is less debate about anger as a trigger of pain.

26.6.5 Anger as Exacerbating Factor

Though psychological factors may not be instrumental in causing medical conditions, such as diabetes and infectious diseases, they may exacerbate existing physical symptoms. There is little doubt that anger can aggravate the intensity of pre-existing pain. Such exacerbation has already been credited to anxiety, and anger shares with anxiety a similar potential for sympathetic nervous system activation. Preliminary support for this effect comes from Summers, Rapoff, Varghese, Porter, and Palmer (1991) who found that anger-hostility scores explained 33% of the variance in pain severity. A subsequent study showed that internalized anger was the best predictor of pain intensity ratings as well as pain behaviors (Kerns, Rosenberg, & Jacob, 1994). Even when the onset of pain is not due to anger, the intensity of such pre-existing pain can be magnified by anger.

26.6.6 Anger as Perpetuating Factor

Just as anger can intensify pre-existing pain, so can it prolong pre-existing pain. The dependent variable in this case is duration rather than severity. This process may best be referred to as perpetuation. The affective variable in this case is not responsible for pain onset but for the maintenance of pain. This is one of numerous examples of the theory of operant conditioning by which behavior is maintained by its consequences.

Anger may prolong the duration of a pain episode or the chronicity of pain because anger in the pain sufferer often elicits submissive or solicitous responses from others and these responses in turn reinforce pain behaviors (Fordyce, 1976). Greenwood, Thurston, Rumble, Waters, and Keefe (2003) have reviewed studies that show how anger may maintain pain by disrupting work, marital/family harmony, and even relations with health-care providers.

26.6.7 Anger as Consequence

Finally, the predominant type of pain–anger interaction is that anger can be a consequence of pain, just as depression is. The bulk of this treatise has in fact focused on this type of interaction. We have identified the variety of adverse circumstances that attend pain and the corresponding mosaic of targets that elicit anger in the pain sufferer. Clearly this collective outcome is not a reflex but one mediated by a plethora of attributions or appraisals about various wrongdoings or mistreatment and those perceived to be responsible for these circumstances.

26.7 Conclusion

This chapter began with conceptual clarification of pain and anger in addition to a demarcation between these and other related concepts. We then surveyed the many personal, social, and existential challenges that face those in pain. These include the pain itself (which can be personified) plus diagnostic ambiguity; treatment failure; battles with insurance carriers, employers, and the legal system; perceived neglect from significant others; and the notion of being singled out by fate or God. To reiterate, anger hardly occurs in a vacuum or as a reflex of pain but within the context of numerous interactions that are the subject of elaborate interpretation. It is therefore understandable that anger is a consequence of pain. In addition, anger shares other dynamic relationships in which it is far more than just a co-occurrent or correlate of pain. Anger can predispose, precipitate, exacerbate, and/or perpetuate pain. Keeping this manifold framework in mind can assist us in uncovering the bases of pain and suffering and also in developing specific treatments most appropriate to the particular type of interaction between pain and anger.

References

- American Academy of Pain Management. (2006). *Pain is an epidemic: A special message from the director*. Retrieved October 6, 2006, from <http://www.aapmanage.org/literature/Articles/PainAnEpidemic.pdf>
- Berkowitz, L. (1993). Pain and aggression: Some findings and implications. *Motivation and Emotion*, 17, 277–293.
- Berkowitz, L. (1998). Affective aggression: The role of stress, pain, and negative affect. In R. G. Geen & E. Donnerstein (Eds.), *Human aggression: Theories, research, and implications for social policy* (pp. 49–72). San Diego, CA: Academic Press.
- Blumer, D., & Heilbronn, M. (1984). “Chronic pain as a variant of depressive disease”: A rejoinder. *Journal of Nervous and Mental Disease*, 172, 405–407.
- Bulman, R. J., & Wortman, C. B. (1977). Attributions of blame and coping in the “real world”. Severe accident victims react to their lot. *Journal of Personality and Social Psychology*, 35, 351–363.
- Burns, J., Higdon, L., Mullen, J., Lansky, D., & Mei Wei, J. (1999). Relationships among patient hostility, anger expression, depression, and the working alliance in a work hardening program. *Annals of Behavioral Medicine*, 21, 77–82.
- Burns, J. W., Kubilus, A., & Bruehl, S. (2003). Emotion induction moderates effects of anger management style on acute pain sensitivity. *Pain*, 106, 109–118.

- Chapman, S. L., Jamison, R. N., Sanders, S. H., Lyman, D. R., & Lynch, T. (2000). Perceived treatment helpfulness and cost in chronic pain rehabilitation. *Clinical Journal of Pain*, 16, 169–177.
- Connant, L. L. (1998). Psychological variables associated with pain perceptions among individuals with chronic spinal cord injury pain. *Journal of Clinical Psychology in Medical Settings*, 5, 71–90.
- Cordell, W. H., Keene, K. K., Giles, B. K., Jones, J. B., Jones, J. H., & Brizendine, E. J. (2002). The high prevalence of pain in emergency medical care. *American Journal of Emergency Medicine*, 20, 165–169.
- DeGood, D. E., & Kiernan, B. (1996). Perception of fault in patients with chronic pain. *Pain*, 64, 153–159.
- Defrin, R., Ohry, A., Blumen, N., & Urca, G. (2001). Characterization of chronic pain and somatosensory function in spinal cord injury subjects. *Pain*, 89, 253–263.
- Dobson, K. S. (1985). The relationship between anxiety and depression. *Clinical Psychology Review*, 5, 307–324.
- Engel, G. L. (1959). “Psychogenic” pain and the pain prone patient. *American Journal of Medicine*, 26, 899–918.
- Fernandez, E. (1986). A classification system of cognitive coping strategies for pain. *Pain*, 26, 141–151.
- Fernandez, E. (1996). *Pain, anger, and negative emotions*. Presented at the 15th Annual Scientific Meeting of the American Pain Society, Washington, DC.
- Fernandez, E. (1998). The role of affect in somatoform and factitious disorders. *Current Review of Pain*, 2, 109–114.
- Fernandez, E. (2002). *Anxiety, depression, and anger in pain: Research findings and clinical options*. Dallas, TX: Advanced Psychological Resources.
- Fernandez, E. (2003). Anger regulation in childhood. In T. Gullotta & M. Bloom (Eds.), *Encyclopedia of primary prevention and health promotion* (pp. 190–195). New York: Kluwer Academic/Plenum Publishers.
- Fernandez, E. (2005). The relationship between anger and pain. *Current Pain and Headache Reports*, 9, 101–105.
- Fernandez, E. (2008). The angry personality: A representation on six dimensions of anger expression. In G. J. Boyle, D. Matthews, & D. Saklofske (Eds.), *International handbook of personality theory and testing: Vol. 1: Personality theories and models* (402–419). London: Sage.
- Fernandez, E., Clark, T. S., & Rudick-Davis, D. (1999). A framework for conceptualization and assessment of affective disturbance in pain. In A. R. Block, E. F. Kremer, & E. Fernandez (Eds.), *Handbook of pain syndromes: Biopsychosocial perspectives* (pp. 123–147). Mahwah, NJ: Erlbaum.
- Fernandez, E., & Kerns, R. D. (2008). Anxiety, depression, and anger: The core of negative affect in medical populations. In G. J. Boyle, D. Matthews, & D. Saklofske (Eds.), *International handbook of personality theory and testing: Vol. 1: Personality theories and models* (pp. 659–676). London: Sage.
- Fernandez, E., Salinas, N., Swift, P., Iglesias, B., & Towery, S. (1995). *Psychosocial factors that predict anger in chronic pain sufferers*. Sixteenth Annual Scientific Meeting of the Society of Behavioral Medicine, San Diego.
- Fernandez, E., & Turk, D. C. (1995). The scope and significance of anger in the experience of chronic pain. *Pain*, 61, 165–175.
- Fishbain, D. A., Goldberg, M., Meagher, B. R., & Steele, R. (1986). Male and female chronic pain categorized by DSM-III psychiatric diagnostic criteria. *Pain*, 26, 181–197.
- Fordyce, W. E. (1976). *Behavioral methods for chronic pain and illness*. St. Louis: Mosby.
- Frijda, N. (1986). *The emotions*. New York: Cambridge University Press.
- Gatchel, R. J. (2005). *Clinical essentials of pain management* (pp. 241–258). Washington, DC: American Psychological Association.
- Gatchel, R. J., & Okifuji, A. (2006). Evidence-based scientific data documenting the treatment and cost-effectiveness of comprehensive pain programs for chronic nonmalignant pain. *The Journal of Pain*, 7, 779–793.
- Gelkopf, M. (1997). Laboratory pain and style of coping with anger. *Journal of Psychology*, 131, 121–124.
- Gliner, J. G., & Compas, B. E. (1999). Self-blame attributions in women with newly diagnosed breast cancer: A prospective study of psychological adjustment. *Health Psychology*, 18, 475–481.
- Grace, V. M. (1995). Problems of communication, diagnosis, and treatment experienced by women using the New Zealand health services for chronic pelvic pain: A quantitative analysis. *Health Care for Women International*, 16, 521–535.
- Greenwood, K. A., Thurston, R., Rumble, M., Waters, S. J., & Keefe, F. J. (2003). Anger and persistent pain: Current status and future directions. *Pain*, 103, 1–5.
- Hall, G. S. (1899). A study of anger. *The American Journal of Psychology*, 10, 516–591.
- Hart, T., Bogner, J. A., Whyte, J., & Polansky, M. (2003). Attribution of blame in accidental and violence-related traumatic brain injury. *Rehabilitation Psychology*, 48, 86–92.
- Harvey, J. H., & Weary, G. (1984). Current issues in attribution theory and research. *Annual Review of Psychology*, 35, 427–459.

- Heider, F. (1958). *The psychology of interpersonal relations*. New York: Wiley.
- International Association for the Study of Pain (1986). Classification of chronic pain: Descriptions of chronic pain syndromes and definitions of pain terms. *Pain*, (Suppl. 3), S1–S226.
- Jensen, M. C., Brant-Zawadzki, M. N., Obuchowski, N., Modic, M. T., Malkasian, D., & Ross, J. S. (1994). Magnetic resonance imaging of the lumbar spine in people without back pain. *The New England journal of medicine*, 331, 69–73.
- Kerns, R. D., Rosenberg, R., & Jacob, M. C. (1994). Anger expression and chronic pain. *Journal of Behavioral Medicine*, 17, 57–67.
- Kristjansdottir, G. K. (1997). The relationships between pains and various discomforts in schoolchildren. *Childhood*, 4, 491–504.
- Kuppens, P., Van Mechelen, I., Smits, D. J. M., & De Boeck, P. (2003). The appraisal basis of anger: Specificity, necessity, and sufficiency of components. *Emotion*, 3, 254–269.
- Lazarus, R. S. (1991). Cognition and motivation in emotion. *American Psychologist*, 46, 352–367.
- Lazarus, R. S. (2000). Cognitive-motivational-relational theory of emotion. In Y. L. Hanin (Ed.), *Emotions in sport* (pp. 39–63). Champaign, IL: Human Kinetics.
- Lewis, M. (2000). The emergence of human emotions. In M. Lewis & J. Haviland-Jones (Eds.), *Handbook of emotions* (pp. 265–280). New York: Guilford Press.
- Magni, G., Marchetti, M., Moreschi, C., Merskey, H., & Luchini, S. R. (1993 May). Chronic musculoskeletal pain and depressive symptoms in the National Health and Nutrition Examination. I. Epidemiologic follow-up study. *Pain*, 53(2), 163–168.
- Marcussen, R. M., & Wolff, H. G. (1949). A formulation of the dynamics of the migraine headache. *Psychosomatic medicine*, 11, 251–256.
- Martin, P. R., & Teoh, H. J. (1999). Effects of visual stimuli and a stressor on head pain. *Headache*, 39, 705–715.
- Materazzo, F., Cathcart, S., & Pritchard, D. (2000). Anger, depression, and coping interactions in headache activity and adjustment: A controlled study. *Journal of Psychosomatic Research*, 49, 69–75.
- McCracken, L. M. (2005). Social context and acceptance of chronic pain: The role of solicitous and punishing responses. *Pain*, 113, 155–159.
- McCracken, L. M., Evon, D., & Karapas, E. T. (2002). Satisfaction with treatment for chronic pain in a specialty service: Preliminary prospective results. *European Journal of Pain*, 6, 387–393.
- McLean, A. S., Clauw, D. J., Abelson, J. L., & Liberzon, I. (2005). The development of persistent pain and psychological morbidity after motor vehicle collision: Integrating the potential role of stress response systems into a biopsychosocial model. *Psychosomatic medicine*, 67, 783–790.
- McParland, J. L., Whyte, A., & Murphy-Black, T. (2005). An exploration of adjustment to pain: Interactions with attribution and individual difference variables. *Psychology and Health*, 20, 19–33.
- National Chronic Pain Outreach Association (2006). Who does chronic pain affect? Retrieved October 6, 2006, from <http://www.chronicpain.org/>
- Okifuji, A., Turk, D. C., & Curran, S. L. (1999). Anger in chronic pain: Investigations of anger targets and intensity. *Journal of Psychosomatic Research*, 47, 1–12.
- Ortony, A., Clore, G., & Collins, A. (1988). *The cognitive structure of emotions*. New York: Cambridge University Press.
- Parkinson, B. (1999). Relations and dissociations between appraisal and emotion ratings of reasonable and unreasonable anger and guilt. *Cognition and Emotion*, 13, 347–385.
- Parkinson, B. (2007). Getting from situations to emotions: Appraisal and other routes. *Emotion*, 7, 21–25.
- Petrie, K. J., Frampton, T., Large, R. G., Moss-Morris, R., Johnson, M., & Meechan, G. (2005). What do patients expect from their first visit to a pain clinic?. *Clinical Journal of Pain*, 21, 297–301.
- Robbins, H., Gatchel, R. J., Noe, C., Gajraj, N., Polatin, P., Deschner, M., et al. (2003). A prospective one-year outcome study of interdisciplinary chronic pain management: Compromising its efficacy by managed care policies. *Anesthesia and analgesia*, 97, 156–162.
- Roseman, I. J. (2004). Appraisals, rather than unpleasantness or muscle movements, are the primary determinants of specific emotions. *Emotion*, 4, 145–150.
- Roth, R. S., Horowitz, K., & Bachman, J. E. (1998). Chronic myofascial pain : Knowledge of diagnosis and satisfaction with treatment. *Archives of Physical Medicine and Rehabilitation*, 79, 966–970.
- Roy, R. (2001). *Social relations and chronic pain*. Dordrecht, the Netherlands: Kluwer Academic Publishers.
- Roy, R. (2002). *Social relations and chronic pain*. New York: Kluwer Academic Publishers.
- Roy, R. (2006). *Chronic pain and family: A clinical perspective*. New York: Springer Science & Business Media.
- Scherer, K. R., Schorr, A., Johnstone, T. (Eds.). (2001). *Appraisal processes in emotion: Theory, methods, research*. New York: Oxford University Press.

- Shaver, K. G. (1970). Defensive attribution: Effects on severity and relevance on the responsibility assigned for an accident. *Journal of Personality and Social Psychology*, 14, 101–113.
- Siemer, M., & Reisenzein, R. (2007). The process of emotion inference. *Emotion*, 7, 1–20.
- Smedslund, J. (1988). *Psycho-logic*. Heidelberg and New York: Springer.
- Smedslund, J. (1992). How shall the concept of anger be defined?. *Theory and Psychology*, 3, 5–34.
- Smith, C. A., & Ellsworth, P. C. (1985). Patterns of cognitive appraisal in emotion. *Journal of Personality and Social Psychology*, 48, 813–838.
- Smith, C. A., & Kirby, L. D. (2004). Appraisal as a pervasive determinant of anger. *Emotion*, 4, 133–138.
- Summers, J. D., Rapoff, M. A., Varghese, G., Porter, K., & Palmer, P. (1991). Psychosocial factors in chronic spinal cord injury pain. *Pain*, 47, 183–189.
- Wasan, A., Fernandez, E., Jamison, R. N., & Bhattacharya, N. (2007). Association of anxiety and depression with reported disease severity in patients undergoing evaluation for chronic rhinosinusitis. *Annals of Otolaryngology, Rhinology, and Laryngology*, 116, 491–497.
- Wasan, A. D., Gudarz, D., & Jamison, R. N. (2005). The association between negative affect and opioid analgesia in patients with discogenic low back pain. *Pain*, 117, 450–461.
- Wasan, A. D., Whooten, J., & Jamison, R. N. (2005). Dealing with difficult patients in your practice. *Regional Anesthesia and Pain Medicine*, 30, 184–192.
- Weiner, B. (1986). *An attributional theory of motivation and emotion*. New York: Springer-Verlag.
- Worchel, S., Cooper, J., Goethals, G. R., & Olson, J. M. (2000). *Social psychology*. Belmont, CA: Wadsworth.
- World Health Organization. (2004). *World Health Organization supports global effort to relieve chronic pain*. Retrieved October 6, 2006, from <http://www.who.int/mediacentre/News/releases/2004>

Chapter 27

Anger and Psychopathology

Raymond W. Novaco

Abstract Anger has semantic, conceptual, and empirical links to psychopathology. It has long been associated with madness, a diseased mind, and behavioral dyscontrol; claims of temporary insanity and the “heat of passion” defense feature anger. As an eruptive and turbulent emotion, anger activates violent behavior among psychiatric patients, before, during, and after hospitalization. Unlike anxiety and depression, there is no diagnostic category for anger, except perhaps intermittent explosive disorder, for which the criterion is aggressive behavior. Being intrinsically related to threat perception, anger is manifested in a wide variety of psychiatric disorders. With the inherent functionality of anger as point of departure, its involvement in adult psychopathology is presented. Anger emerges in conjunction with delusions and command hallucinations in psychotic disorders, the emotional instability attributes of personality disorders, irritability and “attacks” in mood disorders, impulse control disorders, intellectual disabilities, dementia, and exotic cultural-bound syndromes. As anger often results from trauma, it can be salient in PTSD, significantly affecting the severity and course of PTSD symptoms. The central characteristic of anger in the broad context of clinical disorders is dysregulation – its activation, expression, and experience occur without appropriate controls. Cautions against pathologizing an important emotional state are discussed, along with gains being made in anger treatment.

Anger is a turbulent emotion, and its eruptions are often troubling. Since the classical age, anger has been viewed as a mental disturbance and indicative of an unsettled temperament. Lucius Seneca (44/1817), arguably the first anger scholar, endorsed the view of anger as a “short madness” (p. 222). Seneca, as well as Roman and Greek philosopher/historians such as Cicero and Plutarch, sought eradication of anger in the quest for tranquility of mind. Galen, the famous physician to Marcus Aurelius, viewed anger episodes as marked by madness (Galen, 1963). Anger’s generic link to psychopathology is also semantic. Becoming “enraged” suggests being “rabid,” which connotes a diseased state of mind. *Being* angry, *becoming* mad, and *creating* bedlam (echoing the historic asylum) are semantically and metaphorically linked. The thematic history of anger in philosophical, literary, and mythological contexts is traced by Potegal and Novaco in Chapter 2, including the association of anger with insanity.

Vestiges of ancient perspectives of anger as a cauldron of tumultuous forces interwoven with insanity remain. Early texts in psychiatry, such as Krafft-Ebing (1905) and Tuke (1892) designated a condition of “*excandescencia furibunda*” to pertain to the insanity of anger – i.e., the loss of mental

R.W. Novaco (✉)
University of California, Irvine, CA, USA
e-mail: rwnovaco@uci.edu

control or inhibitory central control that was seen to occur during rage episodes. Krafft-Ebing saw the condition as indicative of brain abnormalities caused by biological conditions, trauma, or structural defects (“idiocy”) and stated that “In such conditions the slightest cause leads to the explosive affect of anger, which, owing to continued reproduction of painful thoughts, is maintained at its height” (p. 56). The eruption of anger in a syndrome of temporary insanity or even lasting psychosis is common enough in many cultures and is in fact highly systematized in cases of “wild man” and “amok” syndromes, as observed by anthropologists in Micronesia and other Pacific Island societies and is viewed as psychopathology (e.g., Carr & Tan, 1976; Gaw & Bernstein, 1992). A more temporally proximate and clinically groomed vestige is reflected in Fava’s (1998) notion of “anger attacks,” to be elaborated later in conjunction with affective disorders and impulse control disorders. Not only does such terminology connote being “seized,” but it conjures a pathological/disease entity that then “explains” aggressive behavior and then is suitably “treated” by medication.

Whatever shortcomings such ancient views and residues might have, it is, nevertheless, unmistakable that anger occurs in conjunction with a wide range of psychiatrically classified disorders, including impulse control dysfunctions, mood disorders, many personality disorders, and schizophrenia, especially paranoid schizophrenia. Anger has long been recognized as a feature of clinical conditions that result from trauma, such as dissociative disorders, brain-damage syndromes, and, especially, posttraumatic stress disorder. Anger also appears in mental state disturbances produced by general medical conditions, such as dementia, substance abuse disorders, and neurological dysfunctions resulting from perinatal difficulties. There is now vast literature on anger in psychopathology. This chapter can only provide an overview and will focus on adult psychopathology.

The approach taken here regarding anger and psychopathology is a modest one, which is simply to map anger’s identified involvements in various psychiatric disorder classifications. Unlike for depression and anxiety, there is no set of diagnostic categories for anger, but neither depression nor anxiety is an emotional state. Humans are hardwired for anger because of its survival functions. There can be no sensible thoughts to negate it, much as the Stoics and the Victorians tried. Various authors have advanced the idea of “anger disorders” (DiGuiseppe & Tafrate, 2007; Feindler, 2006; Kassino, 1995), a subject to be discussed in this chapter’s conclusion, but their propositions are not nosologically persuasive. An earlier proposal was made by Thorne (1953). Intermittent explosive disorder (IED) appeared in the *Diagnostic and Statistical Manual* (DSM) III in 1980 and will be addressed in due course. While the Kessler et al. (2006) epidemiological study of IED was presented in news media accounts as being about “anger disorder,” the diagnostic criteria for IED concern aggressive behavior.

The functionality of anger is unmistakable. In the face of adversity, it can mobilize physical and psychological resources, energize behaviors for corrective action, and facilitate perseverance. In the major aggression theories of Berkowitz (1962, 1993), Feshbach (1964, 1971), and Bandura (1973, 1983), respectively, anger arousal was assigned response-energizing, response-motivating, and response-activating functions, and the more recent aggression theory of Anderson and Bushman (2002) alludes to these. In more fully identifying functions, Novaco (1976) sought to move beyond the aggression realm to the clinical arena. The central idea was that the inherent instrumentality of anger and aggression would be an impediment to therapeutic change efforts and that clinical assessment should incorporate ideographic functional analysis of anger patterns. Encapsulating and recasting that earlier formulation, anger can be seen to have the following functions: it *energizes* behavior as a high arousal state, increasing the amplitude of responding and serving to override inhibition; it *focuses* attention on situational elements having threat significance; it *expresses* or communicates negative sentiment, to convey displeasure and to prompt conflict resolution; it *defends* the self by social distancing and fear suppression, and it also defends self-worth by externalizing attributions of blame for misfortune; it *potentiates* a sense of personal control or empowerment,

among social groups as well as individuals; it *instigates* aggressive behavior due to its survival relevance, symbolic linkages, and learned connections; it *signals* information about personal state and situational significance, which is relevant to self-monitoring; and it *dramatizes* a social role enactment, in the sense of anger expression as dramaturgy played out in accord with social scripts or social rules.

Given the functionality of anger, what demarcate anger dysregulation are the parameters of frequency, reactivity, intensity, duration, and mode of expression. These parameters have different contours in the various forms of psychopathology, and at least one clinically oriented anger assessment instrument (Novaco, 2003) is designed to measure them to provide for treatment formulation. These anger response features can be noted in the presentation of disorders to be given here. Nevertheless, in the clinical context, one should not lose sight of anger as part of the human fabric, magnificently reflected in the autobiography of Clifford Beers (1908), for whom anger was central to his recovery from a debilitating disorder while in a psychiatric hospital.

27.1 Current Contextual Background

At the outset it should be recognized that among hospitalized psychiatric patients in long-term care in both civil commitment and forensic institutions, anger is a salient problem, as identified by clinical staff and by the patients themselves. Importantly, it is linked to assaultive behavior by psychiatric patients both inside and outside such facilities. Anger has been found to be predictive of physical aggression prior to hospital admission (McNeil, Eisner, & Binder, 2003; Novaco, 1994), during institutionalization (Doyle & Dolan, 2006a; Novaco, 1994; Novaco & Taylor, 2004; Wang & Diamond, 1999), and in the community after discharge (Doyle & Dolan, 2006b; Monahan et al., 2001; Skeem, et al. 2006). For example, McNeil et al. (2003) found that patients' self-reported anger was the strongest retrospective predictor of violence prior to their hospitalization, controlling for age, substance-related disorder, bipolar disorder, depressive disorder, and schizophrenia. Regarding violent behavior in hospital, Doyle and Dolan (2006a) reported that anger, both patient-rated and staff-rated, was predictive of physical aggression controlling for age, gender, length of stay, and major mental disorder. Similarly, in the Novaco and Taylor (2004) study, patient-rated anger significantly accounted for patients' assaults in hospital, controlling for age, IQ, length of stay, prior violent offending, and personality variables. For post-discharge community studies, in the landmark MacArthur violence risk project (Monahan et al. 2001), patients assessed as having high anger in hospital were more than twice as likely to be violent in the community at 20 weeks and at 1 year after hospital discharge than were low-anger patients. Most impressively, Skeem et al. (2006), in a post-discharge study with weekly level data and numerous control variables concerning high-risk patients, found a time-ordered relationship between anger and violence the following week – and there was no such relationship for anxiety, depression, psychotic symptoms, or general psychological distress. Among non-hospitalized psychiatric outpatients, Posternak and Zimmerman (2002) found one-fourth of their sample of 1300 to have “extreme levels of anger” (p. 668) in the preceding week.

Within psychiatric facilities, anger and aggression are all too prevalent. Among over 4000 California State Hospital patients, approximately 14% had assaulted someone in hospital in a 30-day period, and approximately 35% were rated by their primary clinician as someone who “gets angry and annoyed easily” (Novaco, 1997). High-anger patients typically have traumatic histories, replete with abandonment and rejection, as well as economic and psychological impoverishment. For them, anger becomes an entrenched mode of reactance to stressful or aversive experiences, and it can underpin inertia against therapy programs. Chronically angry people are reluctant to surrender

the anger-aggression system that they have found useful to engage, partly because they discount the costs of its engagement. Psychiatric staff, in acute admission facilities and in long-term institutions, have stressful occupations due to the anger episodes of the patients in their care. Posttraumatic stress disorder commonly occurs among hospital staff victims of patients' assault (e.g., Caldwell, 1992; Wykes & Whittington, 1998).

With this backdrop, anger in psychiatric disorders is here examined, beginning with psychotic disorders, then mood and anxiety disorders, followed by impulse control and personality disorders, and lastly organically based cognitive impairment disorders. It is opportune to start with psychoses, as this category is most closely tied to insanity and because anger is intrinsic to the "provocation defense" of "temporary insanity," which has surfaced in the context of non-psychosis psychiatric disorders.

27.2 Anger and Psychotic Disorders

Dysregulated anger, as a psychotic state, appears in amok – an eruptive syndrome of frenzied violence in Southeast Asian societies (cf. Spores, 1988). Amok and similar syndromes in other cultures were discussed by Averill (1982) in his landmark book on anger, and Potegal and Novaco (Chapter 1) discussed amok in conjunction with the theme of various warrior cultures, including the Norse "berserkers." Amok is almost exclusively a male syndrome, and informative historical/psychiatric accounts are given by Gaw and Bernstein (1992) and Hatta (1996). The Western cultural parallel is spree murder, where multiple victims are killed in multiple locations, typically over a period of hours in frenzied, impulsive attacks by someone with serious mental disorder.

Amok was viewed as an acute delirium with hallucinatory imaging by Van Loon (1927). Psychotic disorders were diagnosed by Schmidt, Hill, and Guthrie (1977) in the majority of their sample of 24 cases in Borneo. Previously viewed as an "exotic psychosis" (Weidman & Sussex, 1971), amok is technically classified in DSM-IV as a culture-bound syndrome, involving a dissociative, brief psychotic episode; however, the culture-boundedness of an angry homicidal paroxysm is far from evident. Kua (1991), for Malaysians, sees its causation in depression as well as psychoses. Gaw and Bernstein (1992) classify it as an impulse control disorder, and Westermeyer (1972) found psychosis to be rare in the Laos amok cases he examined; however, his cases lacked "frenzied" quality. Carr and Tan (1976) more carefully classified 21 Malay cases and found 7 to have psychotic delusions or hallucination at hospital admission and another 5 were chronically psychotic. The Singapore amok cases presented by Ellis (1893) were clearly psychotic or judged to be insane. Madness is a core characteristic of the ferocity of berserkers, whose altered consciousness and fearlessness in battle was manifest in their fighting in a trance-like state and being unaffected by wounds. Hall (1899) took note of their rabid fury, and accounts of their psychotic features are given by Fabing (1956) and Speidel (2002).

Despite knowledge of these culturally infused syndromes and of the historical and philosophical background discussed earlier, research on anger among psychotic patients is sparse. A tendency in the psychiatric field to view anger episodes as manifestations of someone's "illness," rather than as a legitimate emotion meriting clinical attention, has perhaps led to this neglect. The absence of an anger-based diagnostic category might also have de-legitimized anger in conceptualizing psychopathology and giving attention to its role in psychoses.

Perhaps the first psychological study of anger among psychotic patients was that of Landis, Ferrall, and Page (1936), who compared "normal" (college students) and "abnormal" (mostly schizophrenic patients) samples on anger and fear questionnaire responses. Anger ratings to a

provocation inventory were generally higher for the students. Contemporary college students indeed report anger at levels approaching those of hospital psychiatric patients (e.g., Eckhardt, Kassinove, Tsytarev, & Sukhodolsky, 1995; Novaco, 2003), but patients are found to have higher anger scores in some studies (e.g., Cullari, 1994). Psychiatric hospital studies whose samples have predominantly been comprised of psychotic patients and have concerned anger in its relationship to violence (e.g., Doyle & Dolan, 2006a, b; Kay, Wolkenfeld, & Murrill, 1988; Novaco & Renwick, 1998) have not examined anger in its relationship to psychotic symptoms, except for Kennedy, Kemp, and Dyer (1992) concerning delusions. Anger has been studied as a hospital ward atmosphere variable (e.g., Eklund & Hansson, 1997; Friis, 1986; Rossberg & Friis, 2003), which psychotic patients prefer to have at a low level. Another early study of anger among psychotic patients was that of Miller (1939), which appeared in the first issue of *Psychosomatic Medicine*, along with other classic articles on anger and blood pressure. Miller linked “repressed hostile emotion” and “repressed aggression” to the systolic and diastolic blood pressure of 60 paranoid and 23 other schizophrenic patients, along with 33 depressed patients and 77 controls, with the repressed hostility/anger paranoids and depressives having the highest blood pressure. The concept of repressed anger will be discussed in conjunction with mood disorders.

Delusions and hallucinations are cardinal symptoms of schizophrenia. Anger is most relevant to paranoid schizophrenia, delusional disorder (persecutory type), and schizoaffective disorder. Because anger can be understood as fundamentally linked to threat perceptions, it is a key dynamic in the paranoid features of psychotic disorders. Kennedy et al. (1992) reported on a sample of delusional disorder (“paranoid psychosis”) patients who had committed serious violence. All had persecutory delusions, and anger was a prominent affect both before and during the violent offense in the majority of cases, as was fear. In his classic article on the “paranoid pseudo community” Cameron (1943) construed paranoia as an outcome of inadequate social learning and consequent deficits in social skills that underpin interpersonal communications – high susceptibility to slights, a deprecatory attitude toward the self, and the inability to air suspicions so as to gauge their objectivity are predisposing for anger and hostility. Cameron (1951) argued that a person who is hostile and insecure will attribute hostility to his surroundings and, when he is lacking in skills needed for social validation, will maximize the confirmatory information in the situation. Antagonistic behavior, in turn, elicits rejection and counteraggression from others, which then serves to reinforce paranoid beliefs.

Delusion, anger, and paranoid confirmation bias are given dramatic shape in Shakespeare’s Othello, whose heightened suspiciousness and jealousy (“the green-eyed monster”) induce him to interpret all in accord with his persecutory delusions. Moving from drama to scientific analysis, the case records of 8134 Munich psychiatric inpatients examined by Soyka, Naber, and Volcker (1991) revealed the prevalence of delusions of jealousy to range from 2.5 to 7% across categories of psychotic disorder. Violence is often the subject of investigations on delusional jealousy, but anger has been neglected. Enoch and Trethowan (1967), who proposed the term “Othello syndrome,” do refer to rage in their presentation, but they give no systematic attention to anger dimensions.

One function of anger is defensive social distancing, as identified earlier. The expression of anger (in face, posture, words, and deeds) signifies combat mode, which serves to deter the closing of distance (physical and psychological) by others. Anger maintains distance from those who threaten us, energizes preparedness for counterattack, and defends self-worth by externalizing attributions of blame for misfortune. However, anger is directed by attention, and what receives attention is guided by integrated cognitive structures (schemas), which incorporate rules about environment–behavior relationships – this is essentially one of the important lessons from Seneca (44 1817). What receives attention is a product of the cognitive network that assigns meaning to events and the complex stimuli

that configure them. Pertinent to paranoia, an interpretive bias to infer malevolence is associated with high anger reactivity which feeds reciprocity between threat perception, anger, and aggression. That conjecture (Novaco, 1978, 1979) was supported in comparison group studies by Nasby, Hayden, and DePaulo (1980) with emotionally disturbed boys and by Copello and Tata (1990) with violent offenders in a high-security hospital. That anger potentiates threat interpretations, independent of anxiety, is demonstrated experimentally by Barazzone and Davey (2009).

The social information processing model of Huesmann (1988, 1998), which features aggressive scripts, normative beliefs, and rehearsal, and that of Dodge (Dodge & Coie, 1987; Crick & Dodge, 1994), which highlights hostile attributional bias in cue interpretation and peer responses that influence decision heuristics, have high relevance here. Understanding of persecutory delusions in terms of cognitive biases is perhaps best exemplified by the work of Bentall and his colleagues (e.g., Bentall, Kinderman, & Kaney, 1994), who see such delusions as protections against threats to self-esteem – external attributions for blame are made in response to self-ideal discrepancies in a self-perpetuating motivational loop.

Evidence of attention bias associated with anger has been shown in a number of studies (e.g., Eckhardt & Cohen, 1997; van Honk et al., 2001; Wenzel & Lystad, 2005). Moreover, someone who has been targeted by physical attack will likely show selective attention to anger-related threat cues, as can be seen in the elaborate neurophysiological evidence of Pollak and Tolley-Schell (2003) regarding the attentional processing of physically abused children to angry faces. They conjectured that poorly modulated attentional control during anger displays contributes to the social-cognitive biases found in abused children (see also Pollak & Kistler, 2002).

Both selective attention to angry faces and slower disengagement from angry faces have been found for delusion-prone individuals in an experimental analogue study (Arguedas, Green, Langdon, & Coltheart, 2006). While persons with paranoid schizophrenia and those with subclinical paranoid delusions have been found to be deficient in identifying angry affect, this may be due to over-scrutiny or extended processing of threatening stimuli (Combs, Michael, & Penn, 2006; Green, Williams, & Davidson, 2001). The review by Green and Phillips (2004) concluded that a two-stage process may be at work in delusional information processing of threat: initial vigilance followed by active avoidance to reduce anxiety. One should of course bear in mind that implicit in the notion of threat is potential harm to the subject – an elementary point, but one that is perhaps lost when “angry faces” are used as experimental stimuli.

The relationship between delusions and violence, and prospectively anger, in schizophrenia remains to be untangled. Threat-related symptoms (e.g., Wessely et al., 1993; Taylor et al., 1998; Swanson et al., 2006) and threat/control-override (TCO) symptoms (i.e., thoughts of threat and of personal control being overridden by external agents) have been linked with violence (e.g., Link & Stueve, 1994; Swanson et al., 1996, Link et al., 1998). Teasdale, Silver, and Monahan (2006) uncovered a gender moderation effect for TCO symptoms, which were associated with violence in men by not women. Command hallucinations are also relevant, as Monahan et al. (2001) found that, when voices commanded someone to harm others, the likelihood of perpetrated violence over the subsequent year was increased. The interplay of anger with delusions and command hallucinations would seem intriguing. It is noteworthy that Grisso, Davis, Vesselinov, Appelbaum, and Monahan (2000) found a strong relationship between hospital patients' anger and imagined violence, which was prospectively related to community violence. One dimension of anger disposition is rumination, which is related to anger's focusing and defensive functions (cf. Chapter 22 by M. Potegal, and Chapter 24 by T.M. Tripp and R.J. Bies, this book). When anger is dysregulated in its duration, as prolonged by perceived threat or adversity and locked by a fixed delusional system, imagined violence can thereby be primed. This is germane to deviation amplifying feedback loops among the cognitive, affective, behavioral, and social systems noted by Cameron (1956).

27.2.1 Temporary “Insanity” and the Provocation Defense

Judicial penalties for homicide have been affected by whether the person who has done the killing did so in the “heat of passion.” Whether killing is judged to be manslaughter, as opposed to murder, and thus receives a mitigated sentence is the context for the “provocation doctrine,” which centrally involves extreme anger as a passion that overrides reason and defeats self-control. For the defense to succeed, the loss of self-control must be caused by the provocation – the killing is judged to have resulted from provoking circumstances that have undermined the defendant’s self-control. The anger is viewed as an uncontrolled reaction to wrongdoing or perceived injustice, which may not be confined to the immediate situation. If the action taken is judged to be proportionate and to meet a “reasonable person” test (what a reasonable or ordinary person would have done under these conditions), the anger or rage, as an extreme mental and emotional disturbance, can mitigate moral culpability, although it is not exonerative.

In his seminal account of the provocation doctrine, Ashworth (1976) leads with an Aristotelian idea that when an act proceeds from anger, ascription of malice aforethought is negated. He analyzed the provocation defense with regard to the “reasonable man” test, anger, and proportionality issues and then suggested that the “inability to exercise normal control may be classified as a mental disorder . . .” (p. 317). There is now considerable legal scholarship about the provocation doctrine (“heat of passion” defense in US law and “provocation defence” in English law), which cannot be reviewed here. A succinct account is given in Bonnie, Coughlin, Jeffries, and Low (2004), and there is a key book by Horder (1992) and intricate legal studies analyses on focal aspects – e.g., Gough (1999) on the emotion component, Holton and Shute (2007) on self-control, and Yannoulidis (2005) on involuntariness due to impaired consciousness. Potegal’s (Chapter 22 by M. Potegal, this book) analysis of the temporal trajectory of anger has great relevance here, given the absence of legal guidelines for cooling time. Horder (1992) approached the rationale for the provocation defense as being rooted in principles of justified moral outrage, whereby severe provocation provides a moral warrant for retailing in anger, plus a concession to human fallibility. He concluded by finding fault with the provocation doctrine, either as moral outrage or loss of self-control. However, in his continuing scholarship on this issue, Horder (2005) argues that it should be required that to qualify for mitigation, *fear* for one’s safety should be present as well as anger – which would bear on the issue of delay between the final act of provocation and the fatal act.

Relevant to the present chapter, “hot anger” has been viewed in this legal domain as being partially excusatory for lethal violence, because it constitutes both a mental and an emotional disturbance – i.e., a dissociated outrage approximating insanity. Thorne (1953), in making an early argument for “anger disorders,” gave a temporary insanity characterization of certain types of murders that merit excusal from punishment, such as diffuse blind rage that develops from a paranoid reaction to an acute or chronically aversive situation. Spiegel and Suskind (2000) engagingly describe a 1859 Washington D.C. murder case that had a successful temporary insanity plea based on an “uncontrollable frenzy” creating a “brainstorm.” Considering the present popularity of the concept of “anger attacks” in the literature on depression, one might note that Carroll and Forrester (2005) indeed warn against the prospect of impulsive violent offenses carried out in rage being seen as warranting excupation due to “failed agency” by virtue of a *depressive* illness.

Related to the “blind rage” notion is the concept of “catathymic violence” (Schlesinger, 1996, 2006; Kirshner & Nagel, 1996; Meloy, 2000; Chapter 22 by M. Potegal, this book), which refers to an uncontrollable rage having its genesis in “deep” underlying conflict rooted in early childhood, such as early trauma or abandonment, that has generated intense anger that incubates, intensifies, and eventually finds a target. The resultant violent and often frenzied outburst is thought to produce tension relief. Schlesinger’s (1996) review and discussion of the “catathymic crisis” concept portrays

the anger-laden violence in terms of personality inadequacies, fixation, disintegration of psychological functioning, and potentially temporary insanity. The presence of delusions and a dissociative state could heighten such latter consideration (see also Kirshner & Nagel, 1996), which Schlesinger (1996, 2006) finds unlikely to succeed, particularly with long-standing rumination being viewed by juries as premeditation and with the absence of a diagnosis of psychosis. The insanity case of John Hinckley turned on the issue of his delusions, against the arguments of the prosecution team – Clarke's (1990) extensive analysis makes a strong case that Hinckley (who was not angry at President Reagan) was certainly angry but not insane.

What is curious from a cognitive psychology perspective is that the law and legal studies in this area seem to assume that the activation of anger is ungovernable but that what one does once becoming angry is governable. Should not the issues of "self-control" and "reasonableness" also apply to the activation of anger and more specifically to its intensity and duration, not just to what one does when angry? As well, the underpinning of the provocation doctrine in "moral outrage" falls apart to the extent that it hinges on intense anger being aroused. First, the term "provocation" mixes the offending event and the emotional response evoked by it. Is the grievousness of the offending act to be gauged by the magnitude of the offended person's anger reaction? Second, moral rectification, to the extent that is thought to be exculpatory, need not involve anger, as even Seneca (44/1817) advised – if killing is needed as retribution, one need not get angry to do it. To be sure, what one *does* is what gets a person in trouble, but, if the trouble-doing can be partially excused by virtue of it being accompanied by extreme anger, that then begs the question about the anger activation and the cognitions that affect its intensity and duration parameters.

This overview of anger in psychoses provides a useful lead to addressing its involvement in mood disorders, where the topic of "anger attacks" reverberates the motifs of anger as passion, madness, and disease.

27.3 Anger and Mood Disorders

Anger is often a strong accompaniment to depression (e.g., Brody, Haaga, Kirk, & Solomon, 1999; Koh, Kim, & Park, 2002; Posternak & Zimmerman, 2002), as is "hostility"¹ (e.g., Schless, Mendles, Kipperman, & Cochrane, 1974; Scocco, Meneghel, Caon, Dello Buono, & De Leo, 2001; Yesavage, 1983). "Irritable mood" has long been designated as a diagnostic characteristic of mood disorders, both bipolar and unipolar, and a "hostile" subtype for depression has been proposed (e.g., Overall & Zisook, 1980). Contemporary research on the involvement of anger in depression is perhaps best exemplified by the studies on "anger attacks" by Fava and his colleagues (e.g., Fava, Anderson, & Rosenbloom, 1990; Fava & Rosenbaum, 1998, 1999; Perlis et al., 2004), as well as others (Painuly, Sharan, & Mattoo, 2005; Sayar et al., 2000). This line of research influenced the DSM-IV Text Revision definition of "irritability" in terms of persistent anger and angry outbursts (cf. Benazzi, 2003). Snaith and Taylor (1985), well-grounded in psychology of aggression concepts, trace the anger-in-depression linkage to Bleuler, but note that "irritable mood" did not appear in *Index Medicus* until 1985. There were other important precursors.

The classic psychoanalytic position on aggression is that "outward aggression is an expression of the death instinct in the service of Eros," and that "any restrictions of aggression directed outwards increases self-destruction" (Freud, 1930, p. 66). This postulate underpins the psychodynamic view

¹Investigators have often failed to differentiate the constructs of anger and hostility, which aggression scholars have done since Buss (1961).

of depression as anger turned inward. The idea of retroflected anger or “hostility turned inward” appeared in *Mourning and Melancholia* (Freud, 1917) and was carried forward by Menninger (1938), Storr (1968), and Madow (1972) in conceptualizing the psychopathology of depression and suicide. At the societal level, Grier and Cobbs (1968) portrayed the rage of blacks in precisely these terms. Freud’s view was that the self-reproaches of the melancholiac are really against a loved object but are redirected to the patient’s own ego. He has observed that in mourning and grief, the bereaved person’s unconscious anger toward the deceased was not allowed into consciousness but appears as inverted hostility.²

In Lindemann’s (1944) classic study of bereaved persons, he found irritability and anger to be normal reactions, yet he described “morbid grief reactions” in which the grief process was distorted by intensified hostility, including furious hostility toward specific persons. However, longitudinal research on bereavement by Clayton and Darvish (1979) found that, for those who remain depressed after 13 months, feelings of anger about the death intensified; but the anger was frequently directed at the deceased, the hospital, and the physician – it was seldom self-directed.

Early research regarding hostility directedness, conducted with psychiatric patients, found *both* inwardly *and* outwardly directed hostility associated with depression (Blackburn, 1974; Lyketsos, Blackburn, & Tsiantis, 1978; Schless et al., 1974; Weissman, Klerman, & Paykel, 1971). Likewise, in the Snaith and Taylor (1985) study with mood and anxiety disorder samples, both inward and outward “irritability” (their scale’s items are largely anger) were strongly related to depression. They concluded, though, that outwardly expressed irritability was an independent mood disorder.

More recently, Koh et al. (2002), with multiple anger measures, compared depressive disorder patients with anxiety disorder and somatoform disorder patients with healthy controls. Degree of depression was highly correlated with anger for the patient groups and for the healthy controls. The group-differentiating anger dimension was *anger-out*, and there were no group differences for *anger-in*. Depressive disorder patients had higher *anger-out* (Spielberger, Jacobs, Russell, & Crane, 1983) scores and higher anger/hostility on two other measures. The data of Riley, Treiber, and Woods (1989) show depressed patients to be higher in anger than normal controls on multiple measures, regardless of directedness. Inwardly directed hostility does accompany depressed mood (Newman & Hirt, 1983; Snaith & Taylor, 1985), but, while it declines in response to treatment of depression, outwardly directed hostility remains (Blackburn, 1974; Lyketos, Blackburn, & Tsiantis, 1978). To be sure, suppressed anger has many deleterious effects – it has been robustly associated with elevated blood pressure and sustained hypertension in laboratory, field, and clinical studies (cf. Robins & Novaco, 2000), and it has been linked to depression in headache patients (Materazzo, Cathcart, & Pritchard, 2000). However, the psychoanalytic retroflection thesis is, overall, unsupported.

Depressive episodes often involve psychomotor agitation and brooding. Beyond these demarcating symptoms, Fava et al. (1990) reported on a series of cases with major depressive disorder (MDD) for whom sudden “spells” of anger occurred – thought to resemble panic attacks. Fava et al. (1993) subsequently found, in a study with 127 medication-free outpatients with MDD, that 44% reported having “anger attacks,” and those who did scored significantly higher on psychometric scales of

²Bandura (1973) cogently disputed psychodynamic accounts of aggression, but vestiges of this view of depression as “repressed anger” remain in the notions of anger “direction” and “inwardly directed hostility.” At the outset, “repression” should not be confused with “suppression.” Many investigators conveniently slip from Freudian theory, which stipulates repression, to self-report measures of “introjected hostility” or “anger in.” Freud (1917), in accounting for melancholia, describes the hostility as *unconscious*. The psychoanalytic concept of “repression” signifies denial, rejection, and keeping something out of consciousness (cf. Freud, 1915). Thus, if someone is repressing anger, then they are not aware of it. To the contrary, people who are depressed commonly report anger and do so straightaway. That does not signify repression. Anger suppression or inhibition of anger expression is, in contrast, a viable concept.

hostility. Fava has asserted that approximately one-third of depressed outpatients have anger attacks (Fava, 1998; Fava & Rosenbaum, 1999). A rate of 60% was found by Mammen et al. (1999) at a specialty clinic for pregnant or postpartum women, and this was significantly associated with diagnoses of unipolar depression. Generally, the prevalence of “anger attacks” in depression is higher among males (see also Winkler, Pjrek, & Kasper, 2005). Leaving aside the issues of whether “anger attacks” (1) constitute a phenomenon any different from simply having a strong anger reaction or (2) serve to define mood disorder subtypes (see also Pasquini, Picardi, Biondi, Gaetano, & Morosini, 2004), the association of anger to depression is evident, and those who are depressed readily report anger experiences.

In bipolar disorders, perhaps especially for bipolar II where “irritability” can have a larger role in the hypomania, anger has salient manifestations. Irritability (i.e., the quality of being easily excited to anger) was demarcated as a symptom in manic depression by Mayer-Gross (1937). Benazzi (2003) and Benazzi and Akiskal (2005) reported that major depressive episodes with anger were about 60% for bipolar II disorder patients, compared to about 36% for those with MDD (unipolar) patients. As well, Perlis et al. (2004) compared bipolar and unipolar depression patients, and those with bipolar disorder had significantly more “anger attacks,” even controlling for general hostility and borderline personality features. However, whether anger is manifested in bipolar disorder may be a function of the assessment interval. When Beigel and Murphy (1971) obtained ward behavior observations over a 14-day, medication-free period, staff-rated anger was higher for unipolar depression patients than for matched bipolar patients.

Anger/irritability perhaps someday may be judged to define subtypes among mood disorders, an idea that received early attention from Mayer-Gross (1937). Overall, Hollister, Johnson, and Pennington (1966) in a study of medication response with male veterans, Overall, Goldstein, and Brauzer (1971) in profile classification analyses of 6000 patients, and Overall and Zisook (1980) studying mood and anxiety disorder patients advanced that conjecture. Harmon Jones et al. (2002) report that asymmetrical frontal cortical responses to anger provocation differentiate hypomania/mania from depression.

27.3.1 Anger, Depression, and Violence

The association between depression and anger extends to aggressive behavior and violence. High anger and hostility in domestically violent men is accompanied by depression (Maiuro, Cahn, Vitaliano, Wagner, & Zegree, 1988). Aggression is patently manifest in the self-harming and suicidal behavior of depressed persons and certainly in the behavior of those with bipolar disorders. In the Scocco et al. (2001) large sample of elderly persons in the community, the presence of suicidal feelings was significantly differentiated by the BSI “hostility” scale (which is really an *anger* measure). Controlling for depression, anxiety, health status, marital status, and use of hypnotics, “hostility” was associated with nearly a threefold increase in risk in suicidal feelings. Studies by Hillbrand and his colleagues with forensic patients have demonstrated that self-destructiveness and interpersonal violence coexist, as a substantial number of violent patients alternate between self-harm and attacks on others (Hillbrand, 1995; Hillbrand, Krystal, Sharpe, & Foster, 1994). Yesavage (1983) had found that suicidal and other self-destructive acts by hospitalized depressives did not correlate with degree of depression but were significantly related to hostility, both self-reported and observer-rated.

Regarding whether depressed persons are violent, the landmark MacArthur study of violence risk (Monahan et al., 2001), involving 1100 discharged psychiatric patients in 3 US metropolitan areas, provides persuasive data. Post-discharge violence at 20 weeks and at 1 year varied significantly as

a function of diagnostic category, and the violence rate was highest for persons with a diagnosis of depression. The 1-year prevalence rate of violence was 28.5% for patients with depression, 22.0% for those with bipolar disorder, and 14.8% for those with schizophrenia. However, in the multivariate analyses of violence risk factors, depression was not significant, whereas anger was significant.

The co-occurrence of depression and anger has been established for youth as well (e.g., Brody et al., 1999; Cautin, Overholser, & Goetz, 2001). Anger and depression are associated with suicide risk among adolescent inpatients (Cautin et al., 2001), and each have been linked to violent behavior and delinquency (Blitstein, Murray, Lytle, Birnbaum, & Perry, 2005; Sigfusdottir, Farkas, & Silver, 2004).

In summary, anger coexists with depression and other mood disorders. Persons who are clinically depressed report anger expressiveness as well as anger suppression, they report anger directed at others as well as anger directed at themselves, and they act violently toward others as well as engage in self-harm. The psychoanalytic conjecture that the etiology of depression is the result of *repressed* anger is without empirical foundation. In contrast, anger suppression – the conscious inhibition of anger expression – remains a valuable concept, particularly as it does not always indicate dysfunction.

The interrelationship between anger, depression, and violence merits focused attention, particularly as this admixture has been manifest in recent episodes of mass murder by adults and juveniles in the USA, Canada, and Europe. Depression and suicidality would seem to heighten the risk of violence presented by high anger, as not having a stake in the future can be expected to diminish inhibitory control.

27.4 Anger and Anxiety Disorders

Although “irritability” does appear as a criterion in acute stress disorder and in generalized anxiety disorder (GAD), for the most part, anger does not feature in anxiety disorders, with the exception of posttraumatic stress disorder (PTSD). It is generally thought that anger is more likely to be found in disorders of depression than of anxiety. For social phobia, the relevance of anger would intuitively seem to be for the alarm produced by angry faces (e.g., Mogg, Philippot, & Bradley, 2004), but Erwin, Heimberg, Schneier, and Liebowitz (2003) found elevated anger experience and expression among patients with social phobia compared to non-anxious controls, and high anger scores differentiated treatment non-completers. In a study comparing five anxiety disorder groups (Moscovitch, McCabe, Antony, Rocca, & Swinson, 2008), all anxiety disorder patients had higher scores on anger/hostility/aggression measures than non-clinical controls, except those with a specific phobia. In that study, the highest anger scores occurred for those with panic disorder, and Gould et al. (1996) reported on “anger attacks” for panic disorder patients. Interestingly, Germaine, Goddard, Woods, Charney, and Heninger (1992) observed that anger responses increased in GAD patients in response to a serotonin agonist, which was not observed in other anxiety disorder groups.

Whether anxiety disorder patients have serious anger problems or merely differ from non-clinical samples remains to be ascertained. In comparing obsessive-compulsive disorder (OCD) patients with university student controls, Whiteside and Abramowitz (2005) found no differences in anger expression scores. Moreover, they concluded that the patients’ anger was simply indicative of general distress, rather than as a central component of OCD or dynamically related to its symptoms. In general, the psychometric scores reported in the above studies are not readily indicative of anger dysregulation, nor are the inclusion criteria for “anger attacks” in the study by Gould et al. (1996). In contrast, it is quite a different case for PTSD, especially combat PTSD. Barlow (1991) proposed a

model of emotional disorder that linked anger to anxiety via the stress concept, differentiating anger from fear in terms of the sense of control or mastery. Stress is integral to PTSD, where anger is now receiving substantial attention.

27.4.1 *Anger and PTSD*

Anger has long been identified as a component of traumatic reactions. Freud's writings about affect associated with trauma largely ignored anger,³ aside from writing about retroflected anger in *Mourning and Melancholia*. However, important historical work in the trauma field, such as Lindemann (1944), Grinker and Spiegel (1945), and Kardiner and Spiegel (1947), offered many observations about anger, hostility, and aggression as trauma-linked responses. Grinker and Spiegel described eruptive anger occurring among flight crewmen reacting to the strains of air combat operations and specified anger and aggression as elements of stress disorder. Kardiner and Spiegel identified explosive irritability and unwarranted rage as a stage in a progressive development of incapacitating breakdowns, beginning with poor appetite and carelessness, then irritability and exaggerated reactions of rage, and then culminating in freezing, sleep disturbances, and being terrified of one's own artillery. These works on WWII combat aftereffects were particularly incisive in describing anger symptom patterns that were given more concerted attention by Vietnam era scholars, such as Bourne (1970), Horowitz and Solomon (1975), and Figley (1978). An elaboration of this background on anger and trauma is given in Novaco and Chemtob (1998).

Anger has been found to be predictive of PTSD chronicity, severity, and treatment response with various trauma populations. The recent meta-analysis by Orth and Wieland (2006) demonstrated that anger is substantially associated with PTSD in trauma-exposed adults. Orth and Wieland (2006) found that traumatic events, such as technological disaster, crime victimization, and health trauma, have medium to large effects for the relationship between anger and hostility and PTSD. The largest effect sizes were obtained for those having military war experience.

Anger is strongly associated with combat-related PTSD (Beckham, Moore, & Reynolds, 2000; Biddle, Creamer, Forbes, Elliot, & Devilly, 2002; Castillo, Fallon, C'de Baca, Conforti, & Qualls, 2002; Elhai, Frueh, Gold, Gold, & Hamner, 2000; Novaco & Chemtob, 1998, 2002, 2007; Lasko, Gurvits, Kuhne, Orr, & Pitman, 1994). Anger is the problem most often reported by soldiers with PTSD, their family members, and clinicians (Biddle et al., 2002), and it was found to be the comorbid symptom most predictive of PTSD symptom change following treatment – higher anger being associated with poorer treatment response (Forbes, Creamer, Hawthorne, Allen, & McHugh, 2003). In the context of combat-related PTSD, Chemtob, Novaco, Hamada, Gross, and Smith (1997) posited an anger regulatory deficits model, whereby anger activation is part of a dyscontrol syndrome marked by heightened arousal, hostile appraisal, and antagonistic behavior in response to severe threat.

Regarding non-combat populations, recent studies show anger to be a key long-term symptom resulting from trauma, as found with sexual assault survivors (Feeny, Zoellner, & Foa, 2000), motor vehicle accident victims (Ehlers, Mayou, & Bryant, 1998, 2003; Mayou, Ehlers, & Bryant,

³Freud's most attentive writing on anger was his essay on Michelangelo's statue of Moses, which mesmerized Freud on visits to Rome, in the church of San Pietro in Vincoli (St. Peter in Chains). The essay was done anonymously ("by ***") in 1914 in the journal *Imago*. Freud concluded that it was Michelangelo's intent to portray Moses as having subdued his inner tempest. He renewed that interpretation in a postscript in 1927 (*Standard Edition*, 237–238).

2002), psychiatric patients (Franklin, Posternak, & Zimmerman, 2002), domestic violence victims (Chemtob & Carlson, 2004; Jarvis, Gordon, & Novaco, 2005), political prisoners (Schutzwohl & Maercker, 2000), and refugees (Hinton, Hsia, Um, & Otto, 2003). In the Hinton et al. (2003) study of Cambodian refugees, 58% of PTSD patients met criteria for anger-associated panic attacks, averaging 6.2 anger attacks in the previous month. Violence exposure is also strongly related to anger as a trauma symptom among adolescents (Singer, Anglin, Song, & Lunghofer, 1995).

Beyond the associational linkage of high anger with PTSD, there is evidence that anger is predictive of the development of the disorder. In a longitudinal study of American Legionnaires who had served in Southeast Asia during the Vietnam War, Koenen, Stellman, Stellman, and Sommer (2003) found that anger at Time 1 (1984) was predictive of PTSD at Time 2 (1998), controlling for initial PTSD and for a host of background and psychosocial factors (including social support, alcohol use, and depression). In the Andrews, Brewin, Rose, and Kirk (2000) study of adult victims of violent crime, anger was predictive of PTSD at 1 month and at 6 months post-crime. Similarly, the Feeney et al. (2000) study on female assault victims reported that anger predicted later PTSD severity. In the vehicular accident studies by Ehlers et al. (1998, 2003) and Mayou et al. (2002), anger was predictive of subsequent and successive PTSD diagnoses and severity.

Laboratory-based anger research by Chemtob, Hamada, Roitblat, and Muraoka (1994) showed that combat veterans' heart rate in response to provocation scenes was related to a multiple measure anger factor, and that PTSD-related anger was independent of anxiety and impulsivity. Research on cardiovascular responses by Beckham et al. (2002) found that PTSD veterans had more rapid anger reactivity and larger blood pressure increases in reliving an anger memory, as well as higher blood pressure during recovery and greater covert hostility. In the context of combat-related PTSD, Chemtob et al. (1997) and Novaco and Chemtob (2002) posited an anger regulatory deficits model, whereby anger activation is part of a dyscontrol syndrome marked by heightened arousal, hostile appraisal, and antagonistic behavior in response to severe threat. The model posits that anger intrinsically entails a disposition for aggression, which is a joint product of yoked provocation and inhibitory forces. In combat, anger can be adaptive in energizing attack behaviors and in suppressing fear, but it can also reduce fighting proficiency by impairing the processing of complex information and behavioral control. Outside of combat, reacting with anger is easily maladaptive, particularly when the person fails to regulate its intensity and expression in accordance with socio-environmental conditions and task requirements.

Novaco and Chemtob (2002) in a study of Hawaii VA combat veterans found that anger accounted for over 40% of the variance in scores on the Mississippi Scale for Combat-Related PTSD (minus its anger-related items) above that associated with age, education, and combat exposure. This finding using a psychometric PTSD symptom measure was confirmed by parallel results for structured clinical diagnosis of PTSD. Re-analysis of the National Vietnam Veterans Readjustment Survey data set shows that independently assessed anger is substantially associated with combat-related PTSD, both psychometrically and diagnostically assessed, controlling for age, education, combat exposure, anxiety, and depression and removing anger/irritability from the PTSD criteria (Novaco & Chemtob, 2007).

Thus, anger has been found to have significance across trauma populations for the severity and course of PTSD. Additionally, anger seems to have an important role in PTSD treatment response, perhaps thwarting the effectiveness of treatment (Feeney et al., 2000; Forbes et al., 2003). To be sure, anger/irritability is a diagnostic symptom of PTSD, but the diagnostic category is polythetic, and anger need not be present. High anger in conjunction with PTSD may define an important subtype of the disorder, particularly for those exposed to war trauma.

27.5 Intermittent Explosive Disorder

Perhaps the psychiatric disorder most closely tied to anger is intermittent explosive disorder (IED), which is among the impulse control disorders in the DSM, since its third edition. It replaced “explosive personality” in DSM-II. The diagnostic criteria, through DSM-IV TR, do not designate “anger” per se, but rather stipulate “aggressive episodes” and “aggressive impulses” that the person fails to resist and which result in assault or property damage “grossly out of proportion to any precipitating psychosocial stressor.” There must be several discrete episodes, and other disorder classifications must be ruled out. The omission of anger among the diagnostic criteria has perhaps been intended to assure anchoring in reliable observations. However, the recent epidemiological study by Kessler et al. (2006) incorporated the term “anger attacks” in the diagnostic interview, perhaps due to co-authorship by Fava on this study.

Coccaro and his colleagues have been on the forefront of research on this diagnostic category. Coccaro, Kavoussi, Berman, and Lish (1998) asserted that the IED diagnostic criteria were too restrictive, because the aggression required was too severe and because of the personality disorder rule-outs. Thus, they put forward research criteria to broaden the classification, allowing for verbal aggression and eliminating personality disorder rule-outs, while retaining that for mania, major depression, psychosis, substances, and general medical conditions. However, they do add a marked distress or functional impairment criterion that is not present in DSM. They reported high reliability and substantial construct validity with psychometric measures of aggressiveness and impulsivity (no anger measures), but their discriminative validity results were weak or contrary. When the expanded criteria were applied in a ($N = 253$) community study (Coccaro, Schmidt, Samuels, & Nestadt, 2004), a lifetime rate of 11% was found, but this narrows to 4% with DSM criteria applied. In a large study of psychiatric outpatients ($N = 1300$), Coccaro, Posternak, and Zimmerman (2005) found a lifetime rate of 6.3% and a current rate of 3.1%, adhering to DSM-IV criteria except the personality disorder rule-outs. In a comparative study with persons meeting IED research criteria tested against various DSM-IV diagnostic groups and healthy controls, McCloskey, Berman, Noblett, and Coccaro (2006) found confirmatory results for the IED research criteria group on psychometric measures of anger and aggression and behavioral measures of aggression (laboratory shock procedure).

The prevalence of IED is generally low, and it is more commonly diagnosed among men. An early study by Monopolis and Lion (1983) found it to be made in 2.4% of 840 cases at a university teaching hospital, and they questioned the rigor of the diagnoses. More recently, Grant, Levine, Kim, and Potenza (2005) found a rate of 6.4% among 204 inpatients in similar settings. McElroy (1999) noted that many in her sample who met criteria for IED had lifetime comorbid bipolar disorder and that the anger (irritability/rage) during the aggressive episodes was mixed with depressed mood, euphoria, increased energy, and racing thoughts. In the National Comorbidity Survey that referred to “anger attacks” (Kessler et al., 2006), the 12-month prevalence for IED among 9282 household survey respondents was 3.9% (7.3% lifetime), excluding cases who met lifetime criteria for bipolar disorder; however, exclusions were not made for personality disorders, so these authors did not fulfill the required rule-outs. Mean age of onset was at 14 years, with a cumulative probability plateau at about 30 years. Ortega, Canino, and Alegria (2008), with national data on 2554 Latinos, obtained a lifetime rate of 5.8% and a 12-month rate of 4.1%, but they omitted PD rule-outs. These authors also inserted the term “anger attack” in the procedure description (p. 134).

A domain in which IED has emerged outside of psychiatric facilities is that of “aggressive driving” – as would pertain to court-referred cases. This is exemplified in studies by Galovski and Blanchard (2002), Galovski, Blanchard, and Veazey (2002), and Malta, Blanchard, and Friedenberg (2005). Malta et al. (2005) found that IED was more prevalent among aggressive drivers than among

non-aggressive drivers, but so were anxiety disorders, cluster B personality disorders, and alcohol/substance abuse or dependence, as were lifetime prevalence of ADHD and conduct disorder. Similarly, in Galovski et al. (2002), IED was more prevalent among aggressive drivers than controls, but so was any AXIS I disorder and any AXIS II disorder. When IED and non-IED aggressive drivers were compared by Galovski and Blanchard (2002), there were no significant differences in anger on various STAXI subscales, the Buss-Durkee Irritability subscale, or the Deffenbacher, Oetting, and Lynch (1994) Driving Anger measure subscales. Thus, whether IED differentiates anger among aggressive drivers has yet to be established.

Not uncommonly in the IED literature, one finds assertions saying or suggesting that IED causes violent behavior. As the diagnostic category is defined by behavioral criteria, the tautology should be transparent. The faulty logic proffers a disease entity that drives the aggressive behavior, and transporting or extending the “anger attacks” concept into this domain heightens the illness motif. It would be quite a different matter to say that recurrent episodes of explosive violent behavior, characterized by uncontrolled anger, are a form of psychopathology, as they fit general criteria for abnormality (i.e., unusualness, social deviance, faulty perceptions, significant personal distress, maladaptiveness, and dangerousness), and that unregulated anger (i.e., high in intensity, reactivity, frequency, and combative expression) is a risk factor for this condition.

27.6 Anger and Personality Disorder

Psychiatric diagnostic classifications are predominantly polythetic categories or fuzzy sets (Jablensky, 2005; Livesley, 1986), and there has been long-standing controversy with regard to psychopathology and diagnostic categories (e.g., Follette & Houts, 1996; Widiger & Samuel, 2005). Definitional problems abound with regard to personality disorder and its assessment, particularly as the categorical classifications of diagnostic systems rub against the dimensional approach of personality measurement (cf. Coid, 2003; Samuel & Widiger, 2006; Widiger & Trull, 2007). Comorbidity among personality disorders is well known, as is the comorbidity of personality disorder with AXIS I disorders (cf. Lenzenweger, Lane, Loranger, & Kessler, 2007). It is curious, to say the least, that two people can meet criterion for a particular personality disorder and not share a single symptom (as can occur with obsessive/compulsive personality disorder) or have very different presentations (as can occur with antisocial personality disorder).

Among personality disorders, anger is manifested most prominently in several “cluster B” (dramatic, emotional, erratic) disorders, these being the antisocial, borderline, and narcissistic types. Anger is also a feature of paranoid personality, which is categorized in cluster A (odd, eccentric). This chapter will only address these four types. Discussion of a fuller range of personality disorders with regard to anger can be found in DiGuiseppe and Tafrate (2007).

27.6.1 *Paranoid Personality*

The association of anger with paranoia was discussed earlier with regard to psychoses and Cameron’s (1943, 1951) incisive delineation, but the presence of persistent psychotic symptoms is a rule-out for the personality disorder diagnosis. For example, the paranoid ideation cannot have systematized delusional qualities. The paranoid personality is pervasively mistrustful and suspicious, hypersensitive to slights, prone to attribute malice, unforgiving of insults, and quick to anger and counterattack.

Perhaps the first proposal for an “anger disorders” diagnosis was made by Thorne (1953), in his presentation of 4 cases from a larger set of 21, all of whom displayed paranoid behavior. Regarding his cases (two chronically angry, paranoid businessmen and two homicidal rage murderers with paranoia concerning spousal infidelity), he asserted that “The anger state prevents adequate reality testing both because of the disintegrative influence of the emotional reaction and because of the perceptual distortions involved” (p. 336). He proposed “anger reactions” as a new classification of personality disorders, with the paranoid states being “systematized projections of chronic anger” (p. 339). Thorne’s proposal was taken up by Grant (1954), who presented three cases representing paranoia (albeit reflecting various forms of psychopathology) accompanied by strong anger reactions and echoed the conjecture that the delusional features were anger projections. A more contemporary psychological analysis of paranoid personality would entail social information processing errors. Turkat, Keane, and Thompson-Pope (1990) showed that those with paranoid personality misread ambiguous situations and are very inclined to perceive ambiguous intentions as hostile. Their proneness to respond with anger was viewed as a problem of emotional control as well as hypersensitivity.

27.6.2 Borderline Personality

Emotional instability is a core feature of the borderline personality disorder (BPD). In the International Classification of Diseases (ICD-10), the “borderline type” is a subclassification of emotionally unstable personality disorder, with the other subclass being the “impulsive type.” The term “borderline” alludes to an area between neurosis and psychosis (Stern, 1938). An unstable self-image and unstable social relationships accompany the emotional instability. The unstable personality elements for BPD were developed for DSM-III (Spitzer, Endicott, & Gibbon, 1979), with “inappropriate intense anger or lack of control of anger” being one of nine symptoms. Anger or irritability also plays in the separate affective instability symptom, as well as to impulsive aggression and self-harm that are common in BPD. Ingram (1986) linked the rage of the borderline patient to threat of abandonment and also to “madness.” Psychodynamically, the anger is viewed as an externalizing defense mechanism. Stormy attachments, swinging from idealization to devaluation, are fraught with anger. Linehan’s (1993) cognitive behavioral perspective also gives emphasis to the affective instability feature. Analyzing BPD criteria among psychiatric patients, Sanislow, Grilo, and McGlashan (2000) found disturbed relatedness, behavioral dysregulation, and affective dysregulation factors, with inappropriate anger loading high on the latter, along with efforts to avoid abandonment. Self-mutilation and parasuicide are intended to express anger, most strongly as anger at the self (Brown, Comtois, & Linehan, 2002).

The interlacing of anger with borderline personality organization (BPO) has been a strong theme in the work of Dutton and colleagues on abuse in intimate relationships (e.g., Dutton, Saunders, Starzomski, & Bartholomew, 1994; Dutton & Starzomski, 1993, 1994; Dutton, Starzomski, & Ryan, 1996, Chapter 30 by D.G. Dutton, this book). In a series of systematic studies Dutton has found that the males’ BPO and anger accounted for a large proportion of variance in abuse toward female partners. His conception of the underlying dynamic for anger-driven abuse is insecure, fearful attachment rooted in childhood trauma experiences. He and his colleagues see BPO as a personality representation of a fearful attachment style that drives intimacy anger.

As dysregulated anger is a cardinal symptom of BPD, there is a large literature concerning it. Noteworthy here are two recent studies on the time course of BPD symptoms. In an experimental study by Jacob et al. (2008), female BPD patients were compared to health controls for their response

to an anger induction story, and although these groups did not differ in the intensity of the anger reaction, the BPD patients had prolonged reactions. More persuasively perhaps from an ecological validity standpoint, Zanarini et al. (2007) reported in a study of 290 BPD patients that among 24 symptoms, many symptoms declined sharply over a 10-year follow-up. The symptom that showed the least sharp decline was intense anger, retained by 45% of those who had this index symptom at hospital admission. A closing point on BPD is that the volatility of such patients is highly challenging for therapists, and countertransference issues loom large. McHenry (1994) provides an engaging discussion pertinent to the anger features.

27.6.3 *Antisocial Personality*

From a social problem standpoint, antisocial personality disorder (ASPD) presents the greatest challenges, as its defining characteristics involve harm-doing behavior. The antisocial, “psychopathic,” or “sociopathic” personality is commonly associated with violence. However, one should first understand that if a person has recurrently engaged in aggressive behavior (with juvenile onset), it is quite easy to meet criterion for ASPD, for which only three symptoms are required, including “repeated performing acts that are grounds for arrest,” “impulsivity and failure to plan ahead,” irritability and aggressiveness,” “reckless disregard for safety of self or others,” and “being indifferent to or rationalizing having hurt (another person).” With that diagnostic definitional set, it should be transparent, even without a scientific study of aggression perspective, that it is rather goofy to invoke ASPD as an explanation for individual-level or societal-level violent behavior, yet this is a common tautology.

Weiss, Lamberti, and Blackman (1960) described chronic antisocial offenders as “constantly angry and resentful people” (p. 671). Indeed, high STAXI trait anger and anger-out have been found in conjunction with ASPD (Perdikouri, Rathbone, Huband, & Duggan, 2007). As well, Dutton (1995) has extensively portrayed domestic violence in terms of intimate rage, and his work with colleagues on partner abuse, just discussed with regard to BPD, has also entailed ASPD. The data of Dutton and Starzomski (1994) show that, while 37% of court-referred men and 46% of self-referred men meet criterion for BPD, the corresponding rates for ASPD are 66 and 54%. Dutton (1995) also presents patterns of association between wife assault and narcissistic and passive-aggressive personalities, as well as PTSD. Tweed and Dutton (1998) differentiated subgroups of instrumental and impulsive batterers, with the former being characterized by antisocial and narcissistic attributes.

Intertwined with discussions of ASPD is the concept of psychopathy, which has had an intricate history. Psychopathic personality disorder, one of the foremost topics in forensic psychology, was a term initially used to refer to disorders of personality in general, including depressive, hysterical, and eccentric forms, as well as cold, impulsive (Cleckley, 1941). In 1952, DSM-II introduced the classification of “sociopathic personality disturbance” to highlight that the disorder emerged in the context of social relationships. DSM-III removed that terminology. Attention to psychopathy intensified with the work of Hare, particularly as he developed the Psychopathy Checklist and its revision (PCL-R; Hare, 1991), which bifurcated psychopathy as interpersonal-affective traits (Factor 1) and socially deviant behaviors (Factor 2). Psychopaths are primarily understood as having a constellation of personality traits (e.g., callous, deceitful, manipulative, lacking remorse, shallow emotions, egocentricity, low anxiety) that are thought to be related to criminality and resistance to treatment, but emerging research has instead pointed to the socially deviant behavior facet as accounting for why the PCL-R predicts violent recidivism. For example, research by Skeem and her colleagues (Skeem, Miller, Mulvey, Tiemann, & Monahan, 2005) show that the predictive relationship to violence is due to a higher-order *non-psychopathic* construct of general lack of self-control and antagonism,

including anger and impulsiveness. Skeem and Cooke (in press) provide an incisive analysis of the vast literature and controversies surrounding the concept of psychopathy and an enlightened way forward.⁴

The role of anger in psychopathy remains to be disentangled. Blackburn and Lee-Evans (1985) asserted that “psychopaths as a group may be more distinguished by angry reactions to provocation of an interpersonal kind, rather than by reactions to thwarting or frustration” (p. 99). Sterling and Edelmann (1988) reported that psychopaths were prone to anger in responding to both anxiety and anger scenarios and pointed to their cognitive distortions. In the complex analysis of Hicks and Patrick (2006) regarding negative emotionality and psychopathy, anger/hostility had a differential relationship to PCL-R factors: while inversely related to the interpersonal-affective facet (Factor 1), it was positively and strongly related to the social deviance facet (Factor 2), controlling for emotional distress, fearfulness, and depression. Anger/hostility was the only negative emotionality variable reliably associated with PCL-R scores.

27.6.4 Narcissistic Personality

Narcissistic rage has been portrayed since the Ajax of Sophocles, as infused with qualities of madness (cf. Lansky, 1996). Psychoanalytic thinking about narcissism and anger is traced by Muscatello and Scudellari (2000), who interweave borderline and paranoid structures (cf. Chapter 11 by M. Lewis, this book.) Horowitz and Arthur (1988) expand the range of narcissistic rage to include tyrants and organizational leaders, giving emphasis to self-righteousness. The anger factor identified by Coid (1993) in conjunction with high-security hospital females with BPD (and designated as psychopaths) was also related to their diagnoses of NPD. Such overlap reflects the comorbidity of personality disorders.

At the core of narcissism is entitlement, which is akin to self-righteousness. Bishop and Lane (2002) provide a psychoanalytic analysis of the entitlement dynamic in anger among NPD patients, including how anger in childhood provides impetus for the development of entitlement as it interferes with empathy for others' needs. Perceived injury and envy in narcissism move anger to vindictiveness. Analogue studies with college students arrayed psychometrically (low/high) on narcissism have found high narcissism related to anger disposition and anger reactivity (Papps & O'Carroll, 1998; Rhodewalt & Morf, 1998). Rhodewalt and Morf highlight self-aggrandizing attributional style that drives narcissistic anger as a response to a grandiose self-image. As in the treatment of BDP patients, therapists' skills are challenged in dealing with a narcissistic personality. Bishop and Lane make many insightful observations in this regard.

To summarize, dysregulated anger is a feature in many personality disorders, especially those in cluster B. Personality disorders entail stable, long-duration behavior patterns that are refractory and pervasive. Anger is an important dynamic in the emotional instability and impulsivity one encounters in personality disorders, not only as it represents subject distress, impairs functioning, and activates dangerous behavior (including self-harm), but it also makes for an unsettling display and reciprocally shapes the social world to which the person is subsequently exposed.

⁴Skeem and Cooke invoke classic construct validation principles in asserting that the understanding and diagnosis of psychopathy must be separated from the enterprise of predicting violence. They argue that the PCL-R's predictive accuracy is based not on the core personality traits but the deviant, antisocial behavioral elements. In essence, then, past violent/criminal behavior is associated with future behavior of the same ilk.

27.7 Anger and Intellectual Disabilities (Mental Retardation)

From childhood onward, the life circumstances and psychosocial experiences of people with intellectual disabilities⁵ are conducive to the activation of anger and aggressive behavior. Recurrent thwarting of physical, emotional, and interpersonal needs, as well as cognitive functioning deficits, impairs their psychosocial adjustment, particularly those who reside in custodial settings, where the prevalence rates for physical aggression are approximately 35% (Harris, 1993; Hill & Bruininks, 1984; Sigafoos, Elkins, Kerr, & Atwood, 1994; Smith, Branford, Collacott, Cooper, & McGrother, 1996). In those epidemiological studies conducted in three continents, physically assaultive behavior is thus identified as an area of clinical concern for this patient population, whether they reside in the community or institutions, and anger figures prominently in their emotional distress (cf. Taylor, 2002; Taylor & Novaco, 2005). In a forensic developmental disability hospital, Novaco and Taylor (2004) found physically assaultive behavior for male patients post-admission was 46.5%. Importantly here, the number of physical assaults was significantly related to anger, controlling for age, length of stay, IQ, violent offense history, and personality variables. High turnover rates and burnout are a consequence of staff exposure to the risk of violence in developmental disability services (Attwood & Joachim, 1994), and so their anger and aggression carries heavy costs for systems entrusted with providing security and rehabilitation.

It is only recently that anger is receiving attention in the field of intellectual disabilities, which is predominantly occurring in the UK, despite the pioneering work of Benson in the USA (Benson & Fuchs, 1999; Benson & Ivins, 1992; Benson, Rice, & Miranti, 1986). Recently, the “anger attacks” concept has come into play in this field (Pary, 2006). Much of the work has been treatment evaluation oriented (e.g., Hagiliassis, Gulbenkoglou, DiMarco, Young, & Hudson, 2005; Lindsay et al., 2004; Rose, West, & Clifford, 2000; Taylor, Novaco, Gillmer, Robertson, & Thorne, 2005). Development of population-suited anger assessment procedures has also occurred, including self-report and staff-rated psychometric measures (e.g., Adler & Lindsay, 2007; Novaco & Taylor, 2004; Taylor, Novaco, Guinan, & Street, 2004) and laboratory-type protocols (Woodcock & Rose, 2007).

There are multiple vectors that can heighten anger disposition among persons with intellectual disabilities. Compared to persons with average intellectual functioning, their higher dependency, frequent denial of rights, more isolated living conditions, problems in communication, lack of knowledge about appropriate behavior, physical impairments, and lack of economic independence heighten their vulnerability to anger-eliciting events. Strickler (2001) asserted that persons with intellectual disability have higher risk for child abuse and domestic violence exposure. Epidemiological research on twin pairs (sampled from 1994 to 1995 birth cohorts in England) by Koenen, Moffitt, Caspi, Taylor, and Purcell (2003) demonstrated that domestic violence was uniquely associated with IQ suppression in dose–response relationship. Thus, domestic violence exposure may exacerbate cognitive functioning impairment present at birth. In turn, diminished intellectual competence is associated with the early adoption of aggressive strategies that persist over time and are predictive of lower adult intellectual functioning (Huesmann and Eron, 1984) and criminal behavior (Farrington, 1989).

Parental models of anger and aggression, including physical abuse prevalent in this population, substantially account for the anger and aggressive behavior of such hospitalized patients (Novaco & Taylor, 2008). An ethnographic study by Zetlin and Turner (1985) of young adults with mild mental retardation living in the community found that adolescence is a key period when temper

⁵The DSM diagnostic term, mental retardation, primarily has current use in the USA, while “intellectual disability” has attained more worldwide usage. Also, the term “developmental disability” is more inclusive, as in addition to mental retardation, the concept includes other conditions that do not necessarily involve significant sub-average intellectual functioning, such as autism, epilepsy, and other neurological conditions.

tantrums and violent, destructive behavior surfaced. For 84% of the sample, the antisocial behavior and emotional lability “either had not been evident before that period or had noticeably intensified during the high school years” (Zetlin & Turner, 1985, p. 575). It was during adolescence that their “differentness” became salient, along with the implications of their social identity for their life and well-being. Perceived rejection from peers and parents was a key factor.

For patients with intellectual disabilities, clinical interventions for aggression have commonly been restricted to behavioristic antecedent control and contingency management regimes (e.g., Marcus, Vollmer, Swanson, Roane, & Ringdahl, 2001) or psychotropic medication (see Tyrer et al., 2008). The all too common tendency was to attribute their emotional difficulties and challenging behavior to their disability, rather than to their emotional state or needs. As anger operates as an antecedent variable in the assaultive behavior of people in this diagnostic classification and is an element of their emotional distress, it should have high psychotherapeutic priority. Anger’s expressive, energizing, and potentiating functions can be used to bolster treatment engagement.

27.8 Anger and Dementia

The frequency of anger declines linearly with age (Schieman, 2003) and older adults report less anger than do younger adults in the context of social interactions with family members, established friends, or new friends (Charles & Piazza, 2007). Among psychiatric disorders of aging, anger and aggressive behavior have been sparsely studied, yet they pose salient problems for persons with dementia and their caregivers. In a national epidemiological study of 5776 Medicare patients with Alzheimer’s disease, the prevalence of episodes of “unreasonable anger” was 45% for whites and 55% for blacks and Latinos (Sink, Covinsky, Newcomer, & Yaffe, 2004; cf. Chapter 1 by M. Potegal and G. Stemmler, this book). Patients with dementia appear to have heightened proneness toward aggression (Burns, Jacoby, & Levy, 1990; Hope, Keene, Fairburn, McShane, & Jacoby, 1997; Webster & Grossberg, 1996). Palmstierna and Wistedt (1987) found that the aggressive behavior exhibited by elderly patients with dementia was generally more frequent, although less severe than that displayed by elders with schizophrenia and other mental health problems.

Aggression is the most frequent reason for older people to be referred to specialist mental health services and is the most common reason for placement breakdown and consequent institutionalization (Margo, Robinson, & Corea, 1980; Steele, Rouner, Chase, & Folstein, 1990). Geriatric patients with dementia needing hospitalization exhibit high rates of violent behavior both before and after hospitalization, as Haller, Binder, and McNeil (1989) found at a university hospital: among 52 patients on an acute locked inpatient unit, 23% physically attacked someone during the 2 weeks prior to admission and, during the first 3 days of hospitalization, 10% physically attacked someone. In Sweden, for example, Olafsdottir, Marcusson, and Skoog (2001) identified aggression and irritable mood as salient problems among elderly people being served by primary care facilities. Psychiatric exams given to 500 patients over the age of 70 in primary care centers found that for 32% of the men and 19% of the women aggression was a significant symptom.

The psychosocial and therapeutic milieu of care facilities, in both psychiatric hospitals and nursing homes, is very negatively affected by anger/aggression episodes. Mental health professionals serving older adults are often exposed to physical assault (Astrom, Bucht, Eismann, Norberg, & Saverman, 2002; Bensley et al., 1997). In the Bensley et al. (1997) study of assault injuries among staff at a large state hospital, those working on a geriatric unit sustained more severe injuries than staff on other psychiatric units. As well, older adults in residential care are susceptible to assaults

from other residents. A study of violent incident injuries by Shinoda-Tagawa et al. (2004) of nursing home residents (73.5% of whom were over 70 years of age), involving a large case–control comparison group, found that persons living on an Alzheimer’s disease unit were at significantly higher risk for injury from the violent behavior of another resident. Chrzescijanski, Moyle, and Creed (2007) concluded that staff do not take into account that anger is a legitimate emotion during the disease process. More than half of their study patients became aggressive when staff approached them. The patient rate for unprovoked physical aggression was 44%; physical aggression when approached and unprovoked physical aggression occurred on average once per week per patient. Staff observational rating procedures for the assessment of anger and aggression in psychogeriatric facilities have been developed and validated (Patel & Hope, 1992; Taylor, DuQueno, & Novaco, 2004).

The well-being of the caregivers of dementia patients is also undermined by anger as shown in a longitudinal study by Vitaliano, Russo, Young, Teri, and Maiuro (1991). The anger of caregivers detracted from their resources and intensified caregiver burden/distress over time. Steffen and Berger (2000) found anger intensity during caregiving to vary with the family relationship, with daughters being higher in anger than wives. In that study, anger impairs caregivers’ ability to respond optimally to caregiving challenges, with patient behavioral problems most strongly identified as anger-eliciting.

Happily, the anger of caregivers can be remedied. Coon, Thompson, Steffen, Sorocco, and Gallagher-Thompson (2003) implemented an anger management intervention that reduced anger, hostility, and depression, as well as boosted self-efficacy for managing behavioral problems and controlling upsetting thoughts.

27.9 Concluding Reflections on Anger and Psychopathology

The central characteristic of anger in the broad context of clinical problems is that it is “dysregulated” – its activation, expression, and ongoing experience occur without appropriate controls. Alternatively stated, in such clinical conditions, there is a substantial incongruence between anger engagement and the requirements for optimal functioning, both short term and long term. Since the writings of Charles Darwin, William James, and Walter B. Cannon, anger has been viewed in terms of the engagement of the organism’s survival systems in response to threat and the interplay of cognitive, physiological, and behavioral components. It is an elementary Darwinian notion that the adaptive value of a characteristic is entailed by its fitness for the environment; if the environment changes, that characteristic may lose its adaptive value, and the organism must adjust. Context-inappropriate activation or prolongation of anger, such that it does not facilitate solving problems of adaptation, is dysfunctional, and, when persistent and refractory, it can be viewed as pathology.

Identifying the clinically significant features of a person’s anger can be perplexing, and it requires a differentiated assessment scheme. There are many psychometric instruments for the assessment of anger and hostility, and the review by Eckhardt, Norlander, and Deffenbacher (2004) provides valuable coverage. Most anger scales or inventories have not been developed with clinical populations, and even the Novaco Anger Scale (Novaco, 2003), which was so developed, does not articulate psychopathological deficits nor is it integrated with categories of psychopathology.

Given the normality of anger as a human emotion and the importance of contextual conditions associated with its activation, designating anger as a pathological condition is less than straightforward. Unlike depression or anxiety, anger does not stop you. It has fear-suppression and pain-suppression functions, countering perceived vulnerability or loss of control. However,

anger's intrinsic association with threat perception and the heightened arousal and impulsivity that accompany its activation entrain the impairments found in many mental disorders.

It has been proposed that there be a formal designation of "anger disorders" from the early calls of Thorne (1953) and Barlow (1991) to fuller proposals by Eckhardt and Deffenbacher (1995), Feindler (2006), and Kassino and Tafrate (2002), followed by the elaborated one by DiGuiseppe and Tafrate (2007). Thus far, advocates of formal diagnostic categories for anger have not put forward empirical grounds for their proposition or a coherent nosology, including guidance for differential diagnosis. As this chapter has presented, anger is enmeshed in a wide range of psychiatric disorders, and having a separate diagnostic category would seem to require that it be excised from those other categories. To the extent that the determinants of dysregulated anger reside in pathological conditions that demarcate particular disorders (e.g., delusions, hallucinations, trauma, labile mood, suspiciousness, impaired attachment relationships, prefrontal cortical deficiencies, explosivity, intellectual disability, degenerative brain disease) it is perhaps best understood in the context of those disorders.

Moreover, there are troublesome decisional quandaries associated with the pathologizing of an emotional state that has important energizing, informational, and potentiating functions and that is a fundamental survival mechanism with extensions to freedom representational symbolic structures. Looming large here are issues regarding coercion and control associated with formal diagnoses. Having a certified "anger disorder" might make for smoother billing of services and perhaps for research funding, but it would increase the likelihood that persons already hospitalized, particularly forensic patients, would be further detained and fitted with an additional illness label. The "anger attacks" and IED literature reverberate with that motif.

What follows from illness conceptions is that, all too commonly, the anger exhibited by psychiatric patients is "treated" by psychotropic medication. However, six meta-analyses on the effectiveness of psychotherapy for anger have been published (Beck & Fernandez, 1998; Del Vecchio & O'Leary, 2004; DiGuiseppe & Tafrate, 2003; Edmonson & Conger, 1996; Sukhodolsky, Kassino, & Gorman, 2004; Tafrate, 1995), which overall have found medium to strong effect sizes, indicating that approximately 75% of those receiving anger treatment improved compared to controls (cf. Chapter 28 by E. Fernandez, this book). Cognitive behavioral therapy (CBT) approaches have the greatest efficacy. As well, meta-analytic reviews fail to include case study reports and multiple baseline studies with clinical populations, for whom CBT has produced significant clinical gains.

CBT for anger has been shown to have applicability to a wide range of client populations, including those with intellectual disabilities previously seen to lack sufficient cognitive capacity or "insight" (Taylor & Novaco, 2005). Anger dysregulation is indicative of distress for patients, which extends to families, friends, associates, and care providers. It is eminently useful that self-report of anger is predictive of violent behavior, as this serves to enlist clients in treatment and have clinical managers value that enterprise. Hospitalized patients with long-standing aggression histories and diverse forms of psychopathology can be engaged in anger treatment and have been shown to benefit. While therapeutic mechanisms underlying treatment gains are not clear, nor is their sustainability or generalizability, we still can be fortified in providing remedies for anger dyscontrol by seeking further advances in understanding its embeddedness in diverse psychopathology.

References

- Adler, L., & Lindsay, W. R. (2007). Exploratory factor analysis and convergent validity of the Dundee Provocation Inventory. *Journal of Intellectual and Developmental Disability*, 32, 190–199.
- Anderson, C. A., & Bushman, B. J. (2002). Human aggression. *Annual Review of Psychology*, 53, 27–51.

- Andrews, B., Brewin, C. R., Rose, S., & Kirk, M. (2000). Predicting PTSD symptoms in victims of violent crime: The role of shame, anger, and childhood abuse. *Journal of Abnormal Psychology, 109*, 69–73.
- Arguedas, D., Green, M. J., Langdon, R., & Coltheart, M. (2006). Selective attention to threatening faces in delusion-prone individuals. *Cognitive Neuropsychiatry, 11*, 557–575.
- Ashworth, A. J. (1976). The doctrine of provocation. *Cambridge Law Journal, 35*, 292–320.
- Astrom, S., Bucht, G., Eismann, M., Norberg, A., & Savemann, B. L. (2002). Incidence of violence towards staff caring for the elderly. *Scandinavian Journal of Caring Science, 16*, 66–72.
- Attwood, T., & Joachim, R. (1994). The prevention and management of seriously disruptive behavior in Australia. In N. Bouras (Ed.), *Mental health in mental retardation: Recent advances and practices*. Cambridge: Cambridge University Press.
- Averill, J. R. (1982). *Anger and aggression: An essay on emotion*. New York: Springer Verlag.
- Bandura, A. (1973). *Aggression: A social learning analysis*. Englewood Cliffs, NJ: Prentice Hall.
- Bandura, A. (1983). Psychological mechanisms in aggression. In R. Geen & E. Donnerstein (Eds.), *Aggression: Theoretical and empirical reviews* (pp. 1–40). New York: Academic Press.
- Barazzone, N., & Davey, G. C. L. (2009). Anger potentiates the reporting of threatening interpretations: An experimental study. *Journal of Anxiety Disorders, 23*, 489–495.
- Barlow, D. H. (1991). Disorders of emotion. *Psychological Inquiry, 2*, 58–71.
- Beck, R., & Fernandez, E. (1998). Cognitive-behavioral therapy in the treatment of anger: A meta-analysis. *Cognitive Therapy and Research, 22*, 63–74.
- Beckham, J. C., Moore, S. D., & Reynolds, V. (2000). Interpersonal hostility and violence in Vietnam combat veterans with chronic posttraumatic stress disorder: A review of theoretical models and empirical evidence. *Aggression and Violent Behavior, 5*, 451–466.
- Beckham, J. C., Vrana, S. R., Barefoot, J. C., Feldman, M. E., Fairbank, J., & Moore, S. D. (2002). Magnitude and duration of cardiovascular responses to anger in Vietnam veterans with and without posttraumatic stress disorder. *Journal of Consulting and Clinical Psychology, 70*, 228–234.
- Beers, C. W. (1908). *A mind that found itself*. Garden City, NY: Doubleday.
- Beigel, A., & Murphy, D. L. (1971). Unipolar and bipolar affective illness. *Archives of General Psychiatry, 24*, 215–220.
- Benazzi, F. (2003). Major depressive disorder with anger: A bipolar spectrum disorder?. *Psychotherapy and Psychosomatics, 72*, 300–306.
- Benazzi, F., & Akiskal, H. (2005). Irritable-hostile depression: further validation as a bipolar depressive mixed stated. *Journal of affective disorders, 84*, 197–207.
- Bensley, L., Nelson, N., Kaufman, J., Silverstone, B., Kalat, J., & Shields, J. W. (1997). Injuries due to assaults on psychiatric hospital employees in Washington State. *American Journal of Industrial Medicine, 31*, 92–99.
- Benson, B. A., & Fuchs, C. (1999). Anger-arousing situations and coping responses of aggressive adults with intellectual disability. *Journal of Intellectual and Developmental Disability, 24*, 207–214.
- Benson, B. A., & Ivins, J. (1992). Anger, depression and self-concept in adults with mental retardation. *Journal of Intellectual Disability Research, 36*, 169–175.
- Benson, B. A., Rice, C. J., & Miranti, S. V. (1986). Effects of anger management training with mentally retarded adults in group treatment. *Journal of Consulting and Clinical Psychology, 54*, 728–729.
- Bentall, R. P., Kinderman, P., & Kaney, S. (1994). The self, attributional processes and abnormal beliefs: Towards a model of persecutory delusions. *Behaviour Research and Therapy, 32*, 331–341.
- Berkowitz, L. (1962). *Aggression: A social psychological analysis*. New York: McGraw-Hill.
- Berkowitz, L. (1993). *Aggression: Its causes, consequences, and control*. New York: McGraw-Hill.
- Biddle, D., Creamer, M., Forbes, D., Elliot, P., & Devilly, G. (2002). Self-reported problems: A comparison between PTSD-diagnosed veterans, their spouses, and clinicians. *Behaviour Research and Therapy, 40*, 853–865.
- Bishop, J., & Lane, R. C. (2002). The dynamics and dangers of entitlement. *Psychoanalytic Psychology, 19*, 739–758.
- Blackburn, I. M. (1974). The pattern of hostility in affective illness. *British Journal of Psychiatry, 125*, 141–145.
- Blackburn, R., & Lee-Evans, J. M. (1985). Reactions of primary and secondary psychopaths to anger-evoking situations. *British Journal of Clinical Psychology, 24*, 93–100.
- Blitstein, J. L., Murray, D. M., Lytle, L. A., Birnbaum, A. S., & Perry, C. L. (2005). Predictors of violent behavior in an early adolescent cohort: Similarities and differences across genders. *Health Education & Behavior, 23*, 175–194.
- Bonnie, R. J., Coughlin, A. M., Jeffries, J. C., & Low, P. W. (2004). *Criminal law*. New York: Foundation Press.
- Bourne, P. G. (1970). *Men, stress, and Vietnam*. Boston: Little, Brown.
- Braund, S., & Most, G. W. (2003). *Ancient Anger: Perspectives from Homer to Galen*. Yale Classical Studies (Vol. XXXII). Cambridge: Ambridge University Press.

- Brody, C., Haaga, D., Kirk, L., & Solomon, A. (1999). Anger in people who have recovered from depression and never-depressed people. *The Journal of Nervous and Mental Disease*, 187, 400–405.
- Brown, M. Z., Comtois, K. A., & Linehan, M. M. (2002). reasons for suicide attempts and nonsuicidal self-injury in women with borderline personality disorder. *Journal of Abnormal Psychology*, 111, 198–202.
- Burns, A., Jacoby, R., & Levy, R. (1990). Psychiatric phenomena in Alzheimer's disease. IV: disorders of behaviour. *British Journal of Psychiatry*, 157, 86–94.
- Buss, A. H. (1961). *The psychology of aggression*. New York: John Wiley and Sons.
- Caldwell, M. F. (1992). Incidence of PTSD among staff victims of patient violence. *Hospital and Community Psychiatry*, 43, 838–839.
- Cameron, N. (1943). The paranoid pseudocommunity. *American Journal of Sociology*, 49, 32–38.
- Cameron, N. (1951). Perceptual organization and behavior pathology. In R. R. Blake & G. V. Ramsay (Eds.), *Perception: An approach to personality* (pp 283–30 (6). New York: Ronald Press.
- Carr, J. E., & Tan, E. K. (1976). In search of the true amok: Amok as viewed within the Malay culture. *American Journal of Psychiatry*, 133, 1295–1299.
- Carroll, A., & Forrester, A. (2005). Depressive rage and criminal responsibility. *Psychiatry, Psychology and Law*, 12, 36–41.
- Castillo, D. T., Fallon, S. K., C'de Baca, J., Conforti, K., & Qualls, C. (2002). Anger in PTSD: General psychiatric and gender differences on the BDHI. *Journal of Loss and Trauma*, 7, 119–128.
- Cautin, R. L., Overholser, J. C., & Goetz, P. (2001). Assessment of mode of anger expression in adolescent psychiatric inpatients. *Adolescence*, 36, 163–170.
- Charles, S. T., & Piazza, J. R. (2007). Memories of social interactions: Age differences in emotional intensity. *Psychology and Aging*, 22, 30–309.
- Chemtob, C. M., & Carlson, J. G. (2004). Psychological effects of domestic violence on children and their mothers. *International Journal of Stress Management*, 11, 209–226.
- Chemtob, C. M., Hamada, R. S., Roitblat, H. L., & Muraoka, M. Y. (1994). Anger, impulsivity, and anger control in combat-related posttraumatic stress disorder. *Journal of Consulting and Clinical Psychology*, 62, 827–832.
- Chemtob, C. M., Novaco, R. W., Hamada, R. S., Gross, D. M., & Smith, G. (1997). Anger regulation deficits in combat-related post-traumatic stress disorder. *Journal of Traumatic Stress*, 10, 17–36.
- Chrzescijanski, D., Moyle, W., & Creedy, D. (2007). Reducing dementia-related aggression through a staff education intervention. *Dementia*, 6, 271–286.
- Clarke, J. W. (1990). *On being mad or merely angry: John W. Hinkley, Jr., and other dangerous people*. Princeton, NJ: Princeton University Press.
- Clayton, P. J., & Darvish, H. S. (1979). Course of depressive symptoms following the stress of bereavement. In J. Barrett (Ed.), *Stress and mental disorder*. New York: Raven.
- Cleckley, H. (1941). *The mask of sanity: An attempt to reinterpret the so-called psychopathic personality*. Oxford: Mosby.
- Coccaro, E. F., Kavoussi, R. J., Berman, M. E., & Lish, J. D. (1998). Intermittent explosive disorder-revised: Development, reliability, and validity of research criteria. *Comprehensive Psychiatry*, 39, 368–376.
- Coccaro, E. F., Posternak, M. A., & Zimmerman, M. (2005). Prevalence and features of intermittent explosive disorder in a clinical setting. *Journal of Clinical Psychiatry*, 66, 1221–1227.
- Coccaro, E. F., Schmidt, C. A., Samuels, J. F., & Nestadt, G. (2004). Lifetime and 1-month prevalence rates of intermittent explosive disorder in a community sample. *Journal of Clinical Psychiatry*, 65, 820–824.
- Coid, J. (1993). An affective syndrome in psychopaths with borderline personality disorder. *British Journal of Psychiatry*, 162, 641–650.
- Coid, J. (2003). Epidemiology, public health and the problem of personality disorder. *British Journal of Psychiatry*, 182(Suppl. 2), 3–10.
- Combs, D. R., Michael, C. O., & Penn, D. L. (2006). Paranoia and emotion perception across the continuum. *British Journal of Clinical Psychology*, 45, 19–31.
- Coon, D. W., Thompson, L., Steffen, A., Sorocco, K., & Gallagher-Thompson, D. (2003). Anger and depression management: Psychoeducational skill training interventions for women caregivers of a relative with dementia. *The Gerontologist*, 43, 678–689.
- Copello, A. G., & Tata, P. R. (1990). Violent behaviour and interpretative bias: An experimental study of the resolution of ambiguity in violent offenders. *British Journal of Clinical Psychology*, 29, 417–428.
- Cornell, D. G., Peterson, C. S., & Richards, H. (1999). Anger as a predictor of aggression among incarcerated adolescents. *Journal of Consulting and Clinical Psychology*, 67, 108–115.
- Crick, N. R., & Dodge, K. A. (1994). A review and reformulation of social information-processing mechanisms in children's social adjustment. *Psychological Bulletin*, 115, 74–101.

- Cullari, S. (1994). Levels of anger in psychiatric inpatients and normal subjects. *Psychological Reports*, 75, 1163–1168.
- Deffenbacher, J. L., Oetting, E. R., & Lynch, R. S. (1994). Development of a driving anger scale. *Psychological Reports*, 74, 83–91.
- Del Vecchio, T., & O'Leary, K. D. (2004). Effectiveness of anger treatments for specific anger problems: A meta-analytic review. *Clinical Psychology Review*, 24, 15–34.
- DiGuiseppe, R., & Tafrate, R. C. (2003). Anger treatment for adults: A meta-analytic review. *Clinical psychology: Science and Practice*, 10, 70–84.
- DiGuiseppe, R., & Tafrate, R. C. (2007). *Understanding anger disorders*. New York: Oxford University Press.
- Dodge, K. A., & Coie, J. D. (1987). Social-information processing factors in reactive and proactive aggression in children's peer groups. *Journal of Personality and Social Psychology*, 53, 1146–1158.
- Doyle, M., & Dolan, M. (2006a). Evaluating the validity of anger regulation problems, interpersonal style, and disturbed mental state for predicting inpatient violence. *Behavioral Sciences and the Law*, 24, 783–798.
- Doyle, M., & Dolan, M. (2006b). Predicting community violence from patients discharged from mental health services. *British Journal of Psychiatry*, 189, 520–526.
- Dutton, D. G. (1995). Intimate abusiveness. *Clinical Psychology: Science and Practice*, 2, 207–224.
- Dutton, D. G., Saunders, K., Starzomski, A., & Bartholomew, K. (1994). Intimacy anger and insecure attachment as precursors of abuse in intimate relationships. *Journal of Applied Social Psychology*, 24, 1367–1386.
- Dutton, D. G., & Starzomski, A. (1993). Perpetrator characteristics associated with women's reports of psychological and physical abuse. *Violence & Victims*, 8, 326–335.
- Dutton, D. G., & Starzomski, A. (1994). Psychological differences between court-referred and self-referred wife assaulters. *Criminal Justice and Behavior*, 21, 203–222.
- Dutton, D. G., Starzomski, A., & Ryan, L. (1996). Antecedents of abusive personality and abuse behavior in wife assaulters. *Journal of Family Violence*, 11, 113–132.
- Eckhardt, C. I., & Cohen, D. J. (1997). Attention to anger-relevant and irrelevant stimuli following naturalistic insult. *Personality and Individual Differences*, 23, 619–629.
- Eckhardt, C. I., & Deffenbacher, J. L. (1995). Diagnosis of anger disorders. In H. Kassirnov (Ed.), *Anger disorders: Definition, diagnosis, and treatment* (pp. 27–48). Washington, DC: Taylor & Francis.
- Eckhardt, C. I., Kassirnov, H., Tsytsarev, S. V., & Sukhodolsky, D. G. (1995). A Russian version of the State-Trait Anger Expression Inventory: Preliminary Data. *Journal of Personality Assessment*, 64, 440–455.
- Eckhardt, C., Norlander, B., & Deffenbacher, J. (2004). The assessment of anger and hostility: A critical review. *Aggression and Violent Behavior*, 9, 17–43.
- Edmonson, C. B., & Conger, J. C. (1996). A review of treatment efficacy for individuals with anger problems: Conceptual, assessment, and methodological issues. *Clinical Psychology Review*, 16, 251–275.
- Ehlers, A., Mayou, R. A., & Bryant, B. (1998). Psychological predictors of chronic posttraumatic stress disorder after motor vehicle accidents. *Journal of Abnormal Psychology*, 107, 508–519.
- Ehlers, A., Mayou, R. A., & Bryant, B. (2003). Cognitive predictors of posttraumatic stress disorder in children: results of a prospective longitudinal study. *Behaviour Research and Therapy*, 41, 1–10.
- Eklund, M., & Hansson, L. (1997). Relationships between characteristics of the ward atmosphere and treatment outcome in a psychiatric day-care unit based on occupational therapy. *Acta Psychiatrica Scandinavica*, 95, 329–335.
- Elhai, J. D., Frueh, B. C., Gold, P. B., Gold, S. N., & Hamner, M. B. (2000). Clinical presentations of posttraumatic stress disorder across trauma populations: A comparison of MMPI-2 profiles of combat veterans and adult survivors of child sexual abuse. *The Journal of Nervous and Mental Disease*, 188, 708–713.
- Ellis, W. G. (1893). The amok of the Malays. *Journal of Mental Science*, 39, 325–338.
- Enoch, M. D., & Trethowan, W. H. (1967). *Some uncommon psychiatric syndromes*. Bristol: John Wright & Sons.
- Erwin, B. A., Heimberg, R. G., Schneier, F. R., & Liebowitz, M. R. (2003). Anger experience and expression in social anxiety disorder: Pretreatment profile and predictors of attrition and response to cognitive-behavioral treatment. *Behavior Therapy*, 34, 331–350.
- Fabing, H. D. (1956). On going berserk: A neurochemical inquiry. *American Journal of Psychiatry*, 113, 409–415.
- Farrington, D. P. (1989). Early predictors of adolescent aggression and adult violence. *Violence and Victims*, 4, 79–100.
- Fava, M., Anderson, K., & Rosenbaum, J. F. (1990). Anger attacks: Possible variants of panic and major depressive disorders. *American Journal of Psychiatry*, 147, 867–870.
- Fava, M. (1998). Depression with anger attacks. *Journal of Clinical Psychiatry*, 59(Suppl 18), 18–22.
- Fava, M., & Rosenbaum, J. F. (1998). Anger attacks in depression. *Depression and Anxiety*, 8, 59–63.
- Fava, M., & Rosenbaum, J. (1999). Anger attacks in patients with depression. *Journal of Clinical Psychiatry*, 60, 21–24.

- Fava, M., Rosenbaum, J., & McCarthy, M. (1991). Anger attacks in depressed outpatients and their response to fluoxetine. *Psychopharmacological Bulletin*, 27, 275–279.
- Fava, M., Rosenbaum, J. F., Pava, J. A., McCarthy, M. K., Steingard, R. J., & Bouffides, E. (1993). Anger attacks in unipolar depression, Part 1: Clinical correlates and response to fluoxetine treatment. *American Journal of Psychiatry*, 150, 1158–1163.
- Feeney, N. C., Zoellner, L. A., & Foa, E. B. (2000). Anger, dissociation, and posttraumatic stress disorder among female assault victims. *Journal of Traumatic Stress*, 13, 89–100.
- Feindler, E. L. (2006). *Anger-related disorders: A practitioner's guide to comparative treatments*. New York: Springer.
- Feshbach, S. (1964). The function of aggression and the regulation of aggressive drive. *Psychological Review*, 71, 257–272.
- Feshbach, S. (1971). Dynamics and morality of violence and aggression. *American Psychologist*, 26, 281–292.
- Figley, C. R. (1978). *Stress disorders among Vietnam veterans: Theory research and treatment*. New York: Bruner/Mazel.
- Follette, W. C., & Houts, A. C. (1996). Models of scientific progress and the role of theory and taxonomy development: A case study of the DSM. *Journal of Consulting and Clinical Psychology*, 64, 1120–1132.
- Forbes, D., Creamer, M., Hawthorne, G., Allen, N., & McHugh, T. (2003). Comorbidity as a predictor of symptom change following treatment in combat-related posttraumatic stress disorder. *Journal of Nervous and Mental Disease*, 191, 93–99.
- Forbes, D., Hawthorne, G., Elliott, P., McHugh, T., Biddle, D., Craemer, M., & Novaco, R. W. (2004). The Dimensions of Anger Reactions (DAR) scale as a measure of anger in combat-related posttraumatic stress disorder. *Journal of Traumatic Stress*, 17, 249–256.
- Franklin, C. L., Posternak, M. A., & Zimmerman, M. (2002). The impact of subjective and expressed anger on the functioning of psychiatric outpatients with post-traumatic stress disorder. *Journal of Interpersonal Violence*, 17, 1263–1273.
- Freud, S. (1914). *The Moses of Michelangelo*. *Standard Edition (Strachey)*, 13, 211–238.
- Freud, S. (1915/1963). Instincts and their vicissitudes; Repression. In P. Rieff (Ed.), *General psychological theory*. New York: Collier Books.
- Freud, S. (1917/1963). Mourning and melancholia. In P. Rieff (Ed.), *General psychological theory*. New York: Collier Books.
- Freud, S. (1930/1961). *Civilization and its discontents*. New York: W. W. Norton.
- Friis, S. (1986). Characteristics of a good ward atmosphere. *Acta Psychiatrica Scandinavica*, 74, 469–473.
- Galen, C. (1963). *On the passions and errors of the soul* (P. W. Harkins, Trans.). Columbus, OH: Ohio State University Press.
- Galovski, T., Blanchard, E. B., & Veazey, C. (2002). Intermittent explosive disorder and other psychiatric co-morbidity among court-referred and self-referred aggressive drivers. *Behaviour Research and Therapy*, 40, 641–651.
- Galovski, T., & Blanchard, E. B. (2002). Psychological characteristics of aggressive drivers with and without intermittent explosive disorder. *Behaviour Research and Therapy*, 40, 1157–1168.
- Gaw, A. C., & Bernstein, R. L. (1992). Classification of amok in DSM-IV. *Hospital and Community Psychiatry*, 43, 789–793.
- Germine, M., Goddard, A. W., Woods, S. W., Charney, D. S., & Heninger, G. R. (1992). Anger and anxiety responses to *m*-chlorophenylpiperazine in generalized anxiety disorder. *Biological Psychiatry*, 32, 457–461.
- Gough, S. (1999). Taking the heat out of provocation. *Oxford Journal of Legal Studies*, 19, 481–493.
- Gould, R. A., Ball, S., Kaspi, S. P., Otto, M. W., Pollack, M. K., Shekhar, A., et al. (1996). Prevalence and correlates of anger attacks: A two site study. *Journal of affective disorders*, 39, 31–38.
- Grant, J. E., Levine, L., Kim, D., & Potenza, M. N. (2005). Impulse control disorders in adult psychiatric inpatients. *American Journal of Psychiatry*, 162, 2184–2188.
- Grant, V. W. (1954). Anger reaction in paranoids. *Journal of Clinical Psychology*, 10, 275–279.
- Green, M. J., & Phillips, M. L. (2004). Social threat perception and the evolution of paranoia. *Neuroscience and Biobehavioral Reviews*, 28, 333–342.
- Green, M. J., Williams, L. M. N., & Davidson, D. J. (2001). Processing of threat-related affect is delayed in delusion-prone individuals. *British Journal of Clinical Psychology*, 40, 157–165.
- Grier, W. H., & Cobbs, P. M. (1968). *Black rage*. New York: Basic Books.
- Grinker, R. R., & Spiegel, J. P. (1945). *Men under stress*. Philadelphia: Blackington.
- Grisso, T., Davis, J., Vesselinov, R., Appelbaum, P. S., & Monahan, J. (2000). Violent thoughts and violent behavior following hospitalization for mental disorder. *Journal of Consulting and Clinical Psychology*, 68, 388–398.

- Hagiliassis, N., Gulbenkoglou, H., DiMarco, M., Young, S., & Hudson, A. (2005). The anger management project: A group intervention for anger in people with physical and multiple disabilities. *Journal of Intellectual and Developmental Disability, 30*, 86–96.
- Hall, G. S. (1899). A study of anger. *American Journal of Psychology, 10*, 516–591.
- Haller, E., Binder, R. L., & McNeill, D. E. (1989). Violence in geriatric patients with dementia. *Bulletin of the American Academy of Psychiatry and Law, 17*, 183–188.
- Hare, R. D. (1991). *The Hare Psychopathy Checklist-Revised Manual*. Toronto: Multi-Health Systems.
- Harmon-Jones, E., Abramson, L. Y., Sigelman, J., Bohlig, A., Hogan, M. E., & Harmon-Jones, C. (2002). Proneness to hypomania/mania symptoms or depression symptoms and asymmetrical frontal cortical responses to an anger-evoking event. *Journal of Personality and Social Psychology, 82*, 610–618.
- Harris, P. (1993). The nature and extent of aggressive behaviour amongst people with learning difficulties (mental handicap) in a single health district. *Journal of Intellectual Disability Research, 37*, 221–242.
- Hatta, S. M. (1996). A Malay crosscultural worldview and forensic review of amok. *Australian and New Zealand Journal of Psychiatry, 30*, 505–510.
- Hicks, B. M., & Patrick, C. J. (2006). Psychopathy and negative emotionality: Analyses of suppressor effects reveal distinct relations with emotional distress, fearfulness, and anger-hostility. *Journal of Abnormal Psychology, 115*, 276–287.
- Hill, B. K., & Bruinincks, R. H. (1984). Maladaptive behaviour of mentally retarded individuals in residential facilities. *American Journal of Mental Deficiency, 88*, 380–387.
- Hillbrand, M. (1995). Aggression against self and aggression against others in violent psychiatric patients. *Journal of Consulting and Clinical Psychology, 63*, 668–671.
- Hillbrand, M., Krystal, J. H., Sharpe, K. S., & Foster, H. G. (1994). Clinical predictors of self-mutilation in hospitalized forensic patients. *Journal of Nervous and Mental Disease, 182*, 9–13.
- Hinton, D., Hsia, C., Um, K., & Otto, M. W. (2003). Anger-associated panic attacks in Cambodian refugees with PTSD: a multiple baseline examination of clinical data. *Behaviour Research and Therapy, 41*, 647–654.
- Holton, R., & Shute, S. (2007). Self-control in the modern provocation defence. *Oxford Journal of Legal Studies, 27*, 49–73.
- Hope, R. A., Keene, J., Fairburn, C., McShane, R., & Jacoby, R. (1997). Behavioural changes in dementia: are there behavioural syndromes? *International Journal of Geriatric Psychiatry, 12*, 1074–1078.
- Horder, J. (1992). *Provocation and responsibility*. Oxford: Clarendon Press.
- Horder, J. (2005). Reshaping the subjective element in the provocation defence. *Oxford Journal of Legal Studies, 25*, 123–140.
- Horowitz, M. J., & Arthur, R. J. (1988). Narcissistic rage in leaders: The intersection of individual dynamics and group processes. *International Journal of Social Psychiatry, 34*, 135–141.
- Horowitz, M. J., & Solomon, G. F. (1975). A prediction of delayed stress response syndromes in Vietnam veterans. *Journal of Social Issues, 31*, 67–80.
- Huesmann, L. R. (1988). An information processing model for the development of aggressive behavior. *Aggressive Behavior, 14*, 13–24.
- Huesmann, L. R. (1998). The role of social information processing and cognitive schema in the acquisition and maintenance of habitual aggressive behavior. In R. G. Geen & E. Donnerstein (Eds.), *Human aggression: Theories, research, and implications for social policy*. San Diego, CA: Academic Press.
- Huesmann, L. R., & Eron, L. D. (1984). Cognitive processes and the persistence of aggressive behavior. *Aggressive Behavior, 10*, 243–251.
- Ingram, D. H. (1986). Madness and the borderline. *American Journal of Psychoanalysis, 46*, 187–190.
- Jablensky, A. (2005). Categories, dimensions and prototypes: Critical issues for psychiatric classification. *Psychopathology, 38*, 201–205.
- Jacob, G. A., Guenzler, C., Zimmermann, S., Scheel, C. N., Rusch, N., Leonhart, R., et al. (2008). Time course of anger and other emotions in women with borderline personality disorder: A preliminary study. *Journal of Behavior Therapy, 39*, 391–402.
- Jarvis, K. L., Gordon, E. E., & Novaco, R. W. (2005). Psychological distress of mothers and children in domestic violence emergency shelters. *Journal of Family Violence, 20*, 389–402.
- Kardiner, A., & Spiegel, H. (1947). *War stress and neurotic illness*. New York: Paul B. Hoeber, Inc.
- Karpman, B. (1941). On the need of separating psychopathy into two distinct clinical types: The symptomatic and the idiopathic. *Journal of Criminal Psychopathology, 3*, 112–137.
- Karpman, B. (1959). Contrasting psychodynamics in two types of psychopathic behavior. *Archives of Criminal Psychodynamics, 3*, 69–107.
- Kassinove, H. (1995). *Anger disorders: Definition, diagnosis, and treatment*. Washington, DC: Taylor & Francis.

- Kassinove, H., & Tafrate, R. C. (2002). *Anger management: The complete treatment guidebook for practitioners*. Atascadero, CA: Impact Publishers.
- Kay, S. R., Wolkenfeld, F., & Murrill, L. M. (1988). Profiles of aggression among psychiatric inpatients. II: Covariates and predictors. *The Journal of Nervous and Mental Disease*, 176, 547–557.
- Kemp, S., & Strongman, K. T. (1995). Anger theory and management: A historical analysis. *American Journal of Psychology*, 108, 397–417.
- Kennedy, H. G., Kemp, L. I., & Dyer, D. E. (1992). Fear and anger in delusional (paranoid) disorder: The association with violence. *British Journal of Psychiatry*, 160, 488–492.
- Kessler, R. C., Coccaro, E. F., Fava, M., Jaeger, S., Jin, R., & Walters, E. (2006). The prevalence and correlates of DSM-IV intermittent explosive disorder in the National Comorbidity Survey Replication. *Archives of General Psychiatry*, 63, 669–678.
- Kirshner, D., & Nagel, L. (1996). Catathymic violence, dissociation, and adoption pathology: Implications for the mental status defense. *International Journal of Offender Therapy and Comparative Criminology*, 40, 204–211.
- Koenen, K. C., Moffitt, T. E., Caspi, A., Taylor, A., & Purcell, S. (2003). Domestic violence is associated with environmental suppression of IQ in young children. *Development and Psychopathology*, 15, 297–311.
- Koenen, K. C., Stellman, J. M., Stellman, S. D., & Sommer, J. F. (2003). Risk factors for course of posttraumatic stress disorder among Vietnam veterans: A 14-year follow-up of American Legionnaires. *Journal of Consulting and Clinical Psychology*, 71, 980–986.
- Koh, K. B., Kim, C. H., & Park, J. K. (2002). Predominance of anger in depression disorders compared with anxiety disorders and somatoform disorders. *Journal of Clinical Psychiatry*, 63, 486–492.
- Krafft-Ebing, vR. (1905). *Textbook of insanity* (C. G. Chaddock, Trans.). Philadelphia: F. A. Davis.
- Kua, E. H. (1991). Amok in nineteenth-century British Malaya history. *History of Psychiatry*, 2, 429–436.
- Landis, C., Ferrall, S., & Page, J. (1936). Fear, anger, and disease: Their inter-correlations in normal and abnormal people. *American Journal of Psychology*, 48, 585–597.
- Lansky, M. R. (1996). Shame and suicide in Sophocles' Ajax. *Psychoanalytic Quarterly*, LXV, 761–786.
- Lasko, N. B., Gurvits, T. V., Kuhne, A. A., Orr, S. P., & Pitman, R. K. (1994). Aggression and its correlates in Vietnam veterans with and without chronic posttraumatic stress disorder. *Comprehensive Psychiatry*, 35, 373–381.
- Lenzenweger, M. F., Lane, M. C., Loranger, A. W., & Kessler, R. C. (2007). DSM-IV personality disorders in the National Comorbidity Survey replication. *Biological Psychiatry*, 62, 553–564.
- Lindemann, E. (1944). Symptomology and management of acute grief. *American Journal of Psychiatry*, 101, 141–148.
- Lindsay, W. R., Allan, R., Parry, C., Macleod, F., Cottrell, J., Overend, H., et al. (2004). Anger and aggression in people with intellectual disabilities: Treatment and follow-up of consecutive referrals and a waiting list comparison. *Clinical Psychology and Psychotherapy*, 11, 255–264.
- Linehan, M. M. (1993). *Cognitive-behavioral treatment of borderline personality disorder*. New York: Guilford.
- Link, B. G., & Stueve, A. (1994). Psychotic symptoms and the violent/illegal behavior of mental patients compared to community controls. In J. Monahan and H. J. Steadman (Eds.), *Violence and Mental Disorders*. Chicago: Chicago University Press.
- Link, B. G., Stueve, A., & Phelan, J. (1998). Psychotic symptoms and violent behaviors: Probing the components of “threat/control-override” symptoms. *Social psychiatry and psychiatric Epidemiology*, 33, S55–S60.
- Livesley, W. J. (1986). Trait and behavioral prototypes of personality disorder. *American Journal of Psychiatry*, 143, 728–732.
- Lyketos, G. C., Blackburn, I. M., & Tsiantis, J. (1978). The movement of hostility during recovery from depression. *Psychological medicine*, 8, 145–149.
- Madow, L. (1972). *Anger*. New York: Charles Scribner's Sons.
- Maiuro, R. D., Cahn, T. S., Vitaliano, P. P., Wagner, B. C., & Zegree, J. B. (1988). Anger, hostility, and depression in domestically violent versus generally assaultive men and nonviolent control subjects. *Journal of Consulting and Clinical Psychology*, 56, 17–23.
- Malgady, R. G., Rogler, L. H., & Cortes, D. E. (1996). Cultural expressions of psychiatric symptoms: Idioms of anger among Puerto Ricans. *Psychological Assessment*, 8, 265–268.
- Malta, L. S., Blanchard, E. B., & Friedenberg, B. M. (2005). Psychiatric and behavioural problems in aggressive drivers. *Behaviour Research and Therapy*, 43, 1467–1484.
- Mammen, O. K., Shear, M. K., Pilonis, P. A., Kolko, D. J., Thase, M. E., & Greeno, C. G. (1999). Anger attacks: Correlates and significance of an unrecognized symptom. *Journal of Clinical Psychiatry*, 60, 633–642.
- Marcus, B. A., Vollmer, T. R., Swanson, V., Roane, H. R., & Ringdahl, J. E. (2001). An experimental analysis of aggression. *Behavior Modification*, 25, 189–213.
- Margo, J. L., Robinson, J. R., & Corea, S. (1980). Referrals to a psychiatric service from old people's homes. *British Journal of Psychiatry*, 136, 396–401.

- Materazzo, F., Cathcart, S., & Pritchard, D. (2000). Anger, depression, and coping interactions in headache activity and adjustment: a controlled study. *Journal of Psychosomatic Research*, 49, 69–75.
- Mayer-Gross, W. (1937). Irritability as a symptom in manic-depressives. *Journal of Mental Science*, 83, 61–70.
- Mayou, R. A., Ehlers, A., & Bryant, B. (2002). Posttraumatic stress disorder after motor vehicle accidents: 3-year follow-up of a prospective longitudinal study. *Behaviour Research and Therapy*, 40, 665–675.
- McCloskey, M. S., Berman, M. E., Noblett, K. L., & Coccaro, E. F. (2006). Intermittent explosive disorder-integrated research diagnostic criteria: Convergent and discriminant validity. *Journal of Psychiatric Research*, 40, 231–242.
- McElroy, S. L. (1999). Recognition and treatment of DSM-IV intermittent explosive disorder. *Journal of Clinical Psychiatry*, 60(suppl 15), 12–16.
- McHenry, S. S. (1994). When the therapist needs therapy: Characterological counter transference issues and failures in the treatment of the borderline personality disorder. *Psychotherapy*, 31, 557–570.
- McNeil, D. E., Eisner, J. P., & Binder, R. L. (2003). The relationship between aggressive attributional style and violence by psychiatric patients. *Journal of Consulting and Clinical Psychology*, 71, 399–403.
- Meloy, J. R. (2000). The nature and dynamics of sexual homicide: An integrative review. *Aggression and Violent Behavior*, 5, 1–22.
- Menninger, K. (1938). *Man against himself*. New York: Harcourt.
- Mikalsen, J. D. (1986). Zeus the father and Heracles the son in tragedy. *Transactions of the American Philological Association*, 116, 89–98.
- Miller, M. L. (1939). Blood pressure findings in relation to inhibited aggressions in psychotics. *Psychosomatic medicine*, 1, 162–172.
- Mogg, K., Philippot, P., & Bradley, B. P. (2004). Selective attention to angry faces in clinical social phobia. *Journal of Abnormal Psychology*, 113, 160–165.
- Monahan, J., Steadman, H. J., Silver, E., Appelbaum, P. S., Robbins, P. C., Mulvey, E. P., et al. (2001). *Rethinking risk assessment: The MacArthur study of mental disorder and violence*. Oxford: Oxford University Press.
- Monopolis, S., & Lion, J. R. (1983). Problems in the diagnosis of intermittent explosive disorder. *American Journal of Psychiatry*, 140, 1200–1202.
- Moscovitch, D. A., McCabe, R. E., Antony, M. M., Rocca, L., & Swinson, R. P. (2008). Anger experience and expression across the anxiety disorders. *Depression and Anxiety*, 25, 107–113.
- Muscattello, C. F., & Scudellari, P. (2000). Anger and narcissism: Between the void of being and the hunger for having. *Psychopathology*, 33, 227–232.
- Nasby, W., Hayden, B., & DePaulo, B. M. (1980). Attributional bias among aggressive boys to interpret unambiguous social stimuli as displays of hostility. *Journal of Abnormal Psychology*, 89, 459–468.
- Newman, R. S., & Hirt, M. (1983). The psychoanalytic theory of depression: Symptoms as a function of aggressive wishes and level of articulation. *Journal of Abnormal Psychology*, 92, 42–48.
- Novaco, R. W. (1976). The functions and regulation of the arousal of anger. *American Journal of Psychiatry*, 133, 1124–1128.
- Novaco, R. W. (1978). Anger and coping with stress: Cognitive-behavioral interventions. In J. P. Foreyt & D. Rathjen (Eds.), *Cognitive behavior therapy: Research and application* (pp. 135–173). New York: Plenum Press.
- Novaco, R. W. (1979). The cognitive regulation of anger and stress. In P. Kendall & S. Hollon (Eds.), *Cognitive behavioral interventions: Theory, research, and procedures* (pp. 241–285). New York: Academic Press.
- Novaco, R. W. (1994). Anger as a risk factor for violence among the mentally disordered. In J. Monahan & H. J. Steadman (Eds.), *Violence and mental disorder: Developments in risk assessment. The John D. and Catherine T. MacArthur Foundation series on mental health and development* (pp. 21–59). Chicago, IL: University of Chicago Press.
- Novaco, R. W. (1997). Remediating anger and aggression with violent offenders. *Legal and Criminological Psychology*, 2, 77–88.
- Novaco, R. W. (2003). *The Novaco anger scale and provocation inventory*. Los Angeles: Western Psychological Services.
- Novaco, R. W., & Chemtob, C. M. (1998). Anger and trauma: Conceptualization, assessment, and treatment. In V. M. Follette, J. I. Rusek, & F. R. Abueg (Eds.), *Cognitive behavioral therapies for trauma* (pp. 162–190). New York: Guilford.
- Novaco, R. W., & Chemtob, C. M. (2002). Anger and combat-related posttraumatic stress disorder. *Journal of Traumatic Stress*, 15(2), 123–132.

- Novaco, R. W., & Chemtob, C. M. (2007). Anger and combat-related PTSD in the National Vietnam Veterans Readjustment survey. Paper presented at the meeting of the American psychological Association, San Francisco, CA, August.
- Novaco, R. W., & Renwick, S. (1998). Anger predictors of the assaultiveness of forensic hospital patients. In E. Sanario (Ed.), *Behaviour and cognitive therapy today: Essays in honour of Hans J. Eysenck* (pp. 199–208). Amsterdam: Elsevier Science.
- Novaco, R. W., & Taylor, J. L. (2004). Assessment of anger and aggression in offenders with developmental disabilities. *Psychological Assessment*, 16, 42–50.
- Novaco, R. W., & Taylor, J. L. (2008). Anger and assaultiveness in male forensic patients with developmental disabilities: Links to volatile parents. *Aggressive Behavior*, 34, 380–393.
- Olafsdottir, M., Marcusson, J., & Skoog, I. (2001). Mental disorders among elderly people in primary care: the Linköping study. *Acta Psychiatrica Scandinavica*, 104, 12–18.
- Ortega, A. N., Canino, G., & Alegria, M. (2008). Lifetime and 12-month intermittent explosive disorder in Latinos. *American Journal of Orthopsychiatry*, 78, 133–139.
- Orth, U., & Wieland, E. (2006). Anger, hostility, and posttraumatic stress disorder in trauma-exposed adults: A meta-analysis. *Journal of Consulting and Clinical Psychology*, 74, 698–706.
- Overall, J. E., Goldstein, B. J., & Brauzer, B. (1971). Symptomatic volunteers in psychiatric research. *Journal of Psychiatric Research*, 9, 31–43.
- Overall, J. E., Hollister, L. E., Johnson, M., & Pennington, V. (1966). Nosology of depression and differential response to drugs. *Journal of the American Medical Association*, 195, 162–164.
- Overall, J. E., & Zisook, S. (1980). Diagnosis and the phenomenology of depressive disorders. *Journal of Consulting and Clinical Psychology*, 48, 626–634.
- Painuly, N., Sharan, P., & Mattoo, S. K. (2005). Relationship of anger and anger attacks with depression. *European Archives of Clinical Neuroscience*, 255, 215–222.
- Palmstierna, T., & Wistedt, B. (1987). Staff observation aggression scale: presentation and evaluation. *Acta Psychiatrica Scandinavica*, 76, 657–663.
- Papps, B. P., & O'Carroll, R. E. (1998). Extremes of self-esteem and narcissism and the experience and expression of anger and aggression. *Aggressive Behavior*, 24, 421–438.
- Pary, R. J. (2006). Anger attacks and mood disorders. *Mental Health Aspects of Developmental Disabilities*, 9, 85–89.
- Pasquini, M., Picardi, A., Biondi, M., Gaetano, P., & Morosini, P. (2004). Relevance of anger and irritability in outpatients with major depressive disorder. *Psychopathology*, 37, 155–160.
- Patel, V., & Hope, R. A. (1992). A rating scale for aggressive behaviour in the elderly – the RAGE. *Psychological medicine*, 22, 211–221.
- Perdikouri, M., Rathbone, G., Huband, N., & Duggan, C. (2007). A comparison of adults with antisocial personality traits with and without childhood conduct disorder. *Annals of Clinical Psychiatry*, 19, 17–23.
- Perlis, R. H., Smoller, J. W., Fava, M., Rosenbaum, J. F., Nierenberg, A. A., & Sachs, G. S. (2004). The prevalence and clinical correlates of anger attacks during depressive episodes in bipolar disorder. *Journal of affective disorders*, 79, 291–295.
- Pollak, S. D., & Kistler, D. J. (2002). Early experience in associated with the development of categorical representations for facial expressions of emotion. *Proceedings of the National Academy of Sciences*, 99, 9072–9076.
- Pollak, S. D., & Tolley-Schell, S. A. (2003). Selective attention to facial emotion in physically abused children. *Journal of Abnormal psychology*, 112, 323–338.
- Posternak, M. A., & Zimmerman, M. (2002). Anger and aggression in psychiatric outpatients. *Journal of Clinical Psychiatry*, 63, 665–672.
- Rhodewalt, F., & Morf, C. C. (1998). On self-aggrandizement and anger: A temporal analysis of narcissism and affective reactions to success and failure. *Journal of Personality and Social Psychology*, 74, 672–685.
- Riley, W. T., Treiber, F. A., & Woods, M. G. (1989). Anger and hostility in depression. *The Journal of Nervous and Mental Disease*, 177, 668–674.
- Robins, S., & Novaco, R. W. (2000). Anger control as a health promotion mechanism. In D. I. Mostofsky & D. H. Barlow (Eds.), *The management of anxiety in medical disorders* (pp. 361–377). Boston: Allyn & Bacon.
- Rose, J., West, C., & Clifford, D. (2000). Group interventions for anger in people with intellectual disabilities. *Research in Developmental Disabilities*, 21, 171–181.
- Rossberg, J. I., & Friis, S. (2003). Do the spontaneity and anger and aggression subscales of the Ward Atmosphere Scale form homogeneous dimensions? A cross-sectional study of 54 wards for psychotic patients. *Acta Psychiatrica Scandinavica*, 107, 118–123.
- Sabini, J., & Silver, M. (1996). On the possible non-existence of emotions: The passions. *Journal for the Theory of Social Behaviour*, 26, 375–398.

- Samuel, D. B., & Widiger, T. A. (2006). Clinicians' judgments of clinical utility: A comparison of the DSM-IV and five-factor models. *Journal of Abnormal Psychology, 115*, 298–308.
- Sandbach, F. H. (1975). *The Stoics*. London: Chatto & Windus.
- Sanislow, C. A., Grilo, C. M., & McGlashan, T. H. (2000). Factor analysis of the DSM-III-R borderline personality disorder criteria in psychiatric inpatients. *American Journal of Psychiatry, 157*, 1629–1633.
- Sayar, K., Guzelhan, Y., Solmaz, M., Ozer, O., Ozturk, M., Acar, B., et al. (2000). Anger attacks in depressed Turkish outpatients. *Annals of Clinical Psychiatry, 12*, 213–218.
- Schieman, S. (2003). Socioeconomic status and the frequency of anger across the life course. *Sociological Perspectives, 46*, 207–222.
- Schlesinger, L. B. (1996). The catathymic crisis, 1912-present: A review and clinical study. *Aggression and Violent Behavior, 1*, 307–316.
- Schlesinger, L. B. (2006). Celebrity stalking, homicide, and suicide: A psychological autopsy. *International Journal of Offender Therapy and Comparative Criminology, 50*, 39–46.
- Schless, A. P., Mendles, J., Kipperman, A., & Cochran, C. (1974). Depression and hostility. *Journal of Nervous and Mental Disease, 159*, 91–100.
- Schmidt, K., Hill, L., & Guthrie, G. (1977). Running amok. *International Journal of Social Psychiatry, 23*, 264–274.
- Schutzwahl, M., & Maercher, A. (2000). Anger in former East German political prisoners: Relationship to posttraumatic stress reactions and social support. *Journal of Nervous and Mental Disease, 188*, 483–489.
- Scocco, P., Meneghel, G., Caon, F., Dello Buono, M., & De Leo, D. (2001). Death ideation and its correlates: Survey of an over-65-year-old population. *Journal of Nervous and Mental Disease, 189*, 210–218.
- Seneca, L. (44/1817). *Seneca's morals*. New York: Harper & Brothers.
- Shinoda-Tagawa, T., Leonard, R., Pontikas, J., McDonough, J. E., Allen, D., & Dreyer, P. I. (2004). Resident-to-resident violent incidents in nursing homes. *Journal of the American Medical Association, 291*, 591–598.
- Sigafoos, J., Elkins, J., Kerr, M., & Atwood, T. (1994). A survey of aggressive behaviour among a population of persons with intellectual disabilities in Queensland. *Journal of Intellectual Disability Research, 38*, 369–381.
- Sigfusdottir, I., Farkas, G., & Silver, E. (2004). The role of depressed mood and anger in the relationship between family conflict and delinquent behavior. *Journal of Youth and Adolescence, 33*, 509–522.
- Singer, M. I., Anglin, T. M., Song, L., & Lunghofer, L. (1995). Adolescents' exposure to violence and associated symptoms of psychological trauma. *Journal of the American Medical Association, 273*, 477–482.
- Sink, K. M., Covinsky, K. E., Newcomer, R., & Yaffe, K. (2004). Ethnic differences in the prevalence and pattern of dementia-related behaviors. *Journal of the American Geriatric Society, 52*, 1277–1283.
- Skeem, J. L., & Cooke, D. J. ((in press)). Is criminal behavior a central component of psychopathy? Conceptual directions for resolving the debate. *Psychological Assessment*.
- Skeem, J. L., Miller, J. D., Mulvey, E., Tiemann, J., & Monahan, J. (2005). Using a five-factor lens to explore the relationship between personality traits and violence in psychiatric patients. *Journal of Consulting and Clinical Psychology, 73*, 454–465.
- Skeem, J. L., Schubert, C., Odgers, C., Mulvey, E. P., Gardner, W., & Lidz, C. (2006). Psychiatric symptoms and community violence among high risk patients: A test of the relationship at the weekly level. *Journal of Consulting and Clinical Psychology, 74*, 967–979.
- Smith, S., Branford, D., Collacott, R. A., Cooper, S. A., & McGrother, C. (1996). Prevalence and cluster typology of maladaptive behaviours in a geographically defined population of adults with learning disabilities. *British Journal of Psychiatry, 169*, 219–227.
- Snaith, R. P., & Taylor, C. M. (1985). Irritability: Definition, assessment and associated factors. *British Journal of Psychiatry, 147*, 127–136.
- Soyka, M., Naber, G., & Volcker, A. (1991). Prevalence of delusional jealousy in different psychiatric disorders: An analysis of 93 cases. *British Journal of Psychiatry, 158*, 549–553.
- Speidel, M. P. (2002). Berserks: A history of Indo-European "mad warriors". *Journal of World History, 13*, 253–290.
- Spiegel, A. D., & Suskind, P. B. (2000). Uncontrollable frenzy and a unique temporary insanity plea. *Journal of Community Health, 25*, 157–179.
- Spielberger, C. D., Jacobs, S., Russell, S., & Crane, R. (1983). Assessment of anger: The State-Trait Anger Scale. In J. D. Butcher & C. D. Spielberger (Eds.), *Advances in personality assessment* (Vol. 2. pp. 157–187). Hillsdale, NJ: Lawrence Erlbaum
- Spitzer, R. L., Endicott, J., & Gibbon, M. (1979). Crossing the border into borderline personality and borderline schizophrenia. *Archives of General Psychiatry, 36*, 17–24.
- Spores, J. C. (1988). *Running amok: An historical inquiry*. Athens, OH: Ohio University Monographs in International Studies, Southeast Asia Series, No. 82.

- Steele, C., Rouner, B., Chase, G. A., & Folstein, M. (1990). Psychiatric symptoms and nursing home placement of patients with Alzheimer's disease. *American Journal of Psychiatry*, 147, 1049–1051.
- Steffen, A. M., & Berger, S. (2000). Relationship differences in anger intensity during caregiving-related situations. *Clinical Gerontologist*, 21, 3–19.
- Sterling, S., & Edelmann, R. J. (1988). Reactions to anger and anxiety-provoking events: Psychopathic and non-psychopathic groups compared. *Journal of Clinical Psychology*, 44, 96–100.
- Stern, A. (1938). Psychoanalytic investigation of and therapy in the borderline group of neuroses. *Psychoanalytic Quarterly*, 7, 467–489.
- Storr, A. (1968). *Human aggression*. New York: Bantam Books.
- Strickler, H. L. (2001). Interaction between family violence and mental retardation. *Mental Retardation*, 39, 461–471.
- Sukhodolsky, D. G., Kassirnov, H., & Gorman, B. S. (2004). Cognitive-behavior therapy for anger in children and adolescents: A meta-analysis. *Aggression and Violent Behavior*, 9, 247–269.
- Sullivan, G. R. (1993). Anger and excuse: Reassessing provocation. *Oxford Journal of Legal Studies*, 13, 421–429.
- Swanson, J. W., Borum, R., Swartz, M. S., & Monahan, J. (1996). Psychotic symptoms and disorders and the risk of violent behavior in the community. *Criminal Behavior and Mental Health*, 6, 309–329.
- Swanson, J. W., Swartz, M. S., Van Dorn, R. A., Elbogen, E. B., Wagner, H. R., Rosenheck, R. A., et al. (2006). A national study of violent behavior in persons with schizophrenia. *Archives of General Psychiatry*, 63, 490–499.
- Tafate, R. C. (1995). Evaluation of treatment strategies for adult anger disorders. In H. Kassirnov (Ed.), *Anger disorders: Definition, diagnosis, and treatment* (pp. 109–129). Washington, DC: Taylor and Francis.
- Taylor, J. L. (2002). A review of the assessment and treatment of anger and aggression in offenders with intellectual disabilities. *Journal of Intellectual Disability Research*, 46, 57–73.
- Taylor, J. L., DuQueno, L., & Novaco, R. W. (2004). Piloting a Ward Anger Rating Scale for older adults with mental health problems. *Behavioural and Cognitive Psychotherapy*, 32, 467–479.
- Taylor, P. J., Leese, M., Williams, D., Butwell, M., Daly, R., & Larkin, E. (1998). Mental disorder and violence. A special (high security) hospital study. *British Journal of Psychiatry*, 172, 218–226.
- Taylor, J. L., & Novaco, R. W. (2005). *Anger treatment for people with developmental disabilities*. Chichester, England: John Wiley & Sons.
- Taylor, J. L., Novaco, R. W., Gillmer, B. T., Robertson, A., & Thorne, I. (2005). A controlled trial of individual cognitive-behavioural anger treatment for people with intellectual disabilities and histories of aggression. *British Journal of Clinical Psychology*, 44, 367–382.
- Taylor, J. L., Novaco, R. W., Guinan, C., & Street, N. (2004). Development of an imaginal provocation test to evaluate treatment for anger problems in people with intellectual disabilities. *Clinical Psychology and Psychotherapy*, 11, 233–246.
- Teasdale, B., Silver, E., & Monahan, J. (2006). Gender, threat/control-override delusions and violence. *Law and Human Behavior*, 30, 649–658.
- Thorne, F. C. (1953). The frustration-anger-hostility states: A new diagnostic classification. *Journal of Clinical Psychology*, 9, 334–339.
- Tuke, D. H. (1892). *A dictionary of psychological medicine*. London: J. & A. Churchill.
- Turkat, I. D., Keane, S. P., & Thompson-Pope, S. K. (1990). Social processing errors among paranoid personalities. *Journal of Psychopathology and Behavioral Assessment*, 12, 263–269.
- Tweed, R. G., & Dutton, D. G. (1998). A comparison of impulsive and instrumental subgroups of batterers. *Violence and Victims*, 13, 217–230.
- Tyrer, P., Oliver-Africano, P. C., Ahmed, Z., Bouras, N., Cooray, S., Deb, S., et al. (2008). Risperidone, haloperidol, and placebo in the treatment of aggressive challenging behaviour in patients with intellectual disability: a randomised controlled trial. *The Lancet*, 371, 57–63.
- van Honk, J., Tuiten, A., van den Hout, M., Putnam, P., de Haan, E., & Stam, H. (2001). Selective attention to unmasked and masked threatening words: relationships to trait anger and anxiety. *Personality and Individual Differences*, 30, 711–720.
- Van Loon, F. H. G. (1927). Amok and latta. *Journal of Abnormal and Social Psychology*, 21, 434–444.
- Vitaliano, P. P., Russo, J., Young, H. M., Tewri, L., & Maiuro, R. D. (1991). Predictors of burden in spouse caregivers of individuals with Alzheimer's disease. *Psychology and Aging*, 6, 392–402.
- Wang, E. W., & Diamond, P. M. (1999). Empirically identifying factors related to violence risk in corrections. *Behavior Sciences and the Law*, 17, 377–389.
- Webster, J., & Grossberg, G. T. (1996). Disinhibition, apathy, indifference, fatigability, complaining & negativism. *International Psychogeriatrics*, 8(Suppl. 3), 403–407.

- Weidman, H. H., & Sussex, J. N. (1971). Cultural values and ego functioning in relation to the atypical culture-bound reactive syndromes. *International Journal of Social Psychiatry*, 17, 83–100.
- Weiss, J., Lambert, J., & Blackman, N. (1960). The sudden murderer. *Archives of General Psychiatry*, 2, 669–678.
- Weissman, M. M., Klerman, G. L., & Paykel, E. S. (1971). Clinical evaluation of hostility in depression. *American Journal of Psychiatry*, 128, 261–266.
- Wenzel, A., & Lystad, C. (2005). Interpretation biases in angry and anxious individuals. *Behaviour Research and Therapy*, 43, 1045–1054.
- Wessely, S., Buchanan, A., Reed, A., Cutting, J., Everitt, P., Garety, P., et al. (1993). Acting on delusions: I. Prevalence. *British Journal of Psychiatry*, 163, 69–76.
- Westermeyer, J. (1972). A comparison of amok and other homicide in Laos. *American Journal of Psychiatry*, 129, 79–85.
- Whiteside, S. P., & Abramowitz, J. S. (2005). The expression of anger and its relationship to symptoms and cognitions in obsessive-compulsive disorder. *Depression and Anxiety*, 21, 106–111.
- Widiger, T. A., & Samuel, D. B. (2005). Diagnostic categories or dimensions? A question for the *Diagnostic and Statistical Manual of Mental Disorders – Fifth Edition*. *Journal of Abnormal Psychology*, 114, 494–504.
- Widiger, T. A., & Trull, T. J. (2007). Plate tectonics in the classification of personality disorder. *American Journal of Psychology*, 62, 71–83.
- Winkler, D., Pjrek, E., & Kasper, S. (2005). Anger attacks in depression – Evidence for a male depressive syndrome. *Psychotherapy and Psychosomatics*, 74, 303–307.
- Woodcock, K. A., & Rose, J. (2007). The relationship between the recognition of facial expressions and self-reported anger in people with intellectual disabilities. *Journal of Applied Research in Intellectual Disabilities*, 20, 279–284.
- Wykes, T., & Whittington, R. (1998). Prevalence and predictors of early traumatic stress reactions in assaulted nurses. *Journal of Forensic Psychiatry*, 9, 643–658.
- Yannoulidis, S. (2005). Excusing fleeting mental states: Provocation, involuntariness and normative practice. *Psychiatry, Psychology and Law*, 12, 23–35.
- Yesavage, J. E. (1983). Direct and indirect hostility and self-destructive behavior by hospitalized depressives. *Acta Psychiatrica Scandinavica*, 68, 345–350.
- Zanarini, M. C., Frankenburg, F. R., Reich, D. B., Silk, K. R., Hudson, J. I., & McSweeney, L. B. (2007). The sub-syndromal phenomenology of borderline personality disorder: A 10-year follow-up study. *American Journal of Psychiatry*, 164, 929–935.
- Zetlin, A. G., & Turner, J. L. (1985). Transition from adolescence to adulthood: Perspectives of mentally retarded individuals and their families. *American Journal of Mental Deficiency*, 89, 570–579.
- Zillmann, D. (1979). *Hostility and aggression*. Hillsdale, NJ: Erlbaum.

Chapter 28

Toward an Integrative Psychotherapy for Maladaptive Anger

Ephrem Fernandez

Abstract This chapter adopts the psychoevolutionary position that anger, like any emotion, is part of a repertoire for handling various adaptational demands. However, it is explained that anger can become maladaptive in terms of the current conceptualization of what constitutes a psychological disorder. In such instances, the treatment options range from philosophico-religious approaches to standalone techniques. Shadowing recent trends in psychotherapy, a case is made for the integration of techniques to regulate anger. As shown, these are sequentially ordered according to phases for prevention, intervention, and postvention of anger. This parallels the course of anger from its onset, through its progression, to its resolution. As illustrated, the prevention phase is primarily behavioral, the intervention phase centers around cognitive strategies, and the postvention phase invokes affective therapies. The resulting cognitive-behavioral affective therapy (CBAT) is programmatic in the additional sense that there is a built-in contingency with which techniques are used. CBAT is more expansive in scope than CBT and in step with the current renaissance of affect as a topic of study within psychology and other disciplines. Preliminary empirical data are reported on the outcome of this program. The large effect sizes obtained are supportive of the absolute and relative efficacy of CBAT in regulating anger. Further research is encouraged for the implementation and evaluation of this integrative program in diverse populations.

28.1 Introduction

Before describing treatments of anger, a couple of relevant issues must be addressed. The first is the justification for treating it. In other words, when is anger maladaptive enough to warrant treatment? Second, what is the target of such treatment? Here, there is a need to distinguish between anger as a primary problem/issue versus epiphenomena of anger or broader constructs merely related to anger. Against this backdrop, it becomes meaningful to present the treatment of anger. In doing so, the purpose of this chapter is not to flesh out individual treatments but to point out some themes underlying past techniques for anger management. This chapter moves toward a novel approach that supplements and integrates past techniques for anger management within a new

E. Fernandez (✉)

Psychology Department, University of Texas at San Antonio, San Antonio, TX, USA

e-mail: ephrem.fernandez@utsa.edu

programmatic format that proceeds from prevention to intervention and finally to remediation or “postvention.”

28.1.1 Why Treat Anger?

Anger has been widely and historically viewed as a problem emotion but these accounts are also interspersed with caveats that not all anger is bad. Hence, the popular adages such as “it is human to get angry,” “there is nothing wrong with anger; it’s what you do with it that makes the difference,” and “there is good anger and there is bad anger.”

From a functionalist standpoint, anger (like any emotion) has adaptive significance. As outlined in Fernandez and Kerns (2008), anger and fear are twin emotions in the defense against aversive stimuli. They subserve what Walter Cannon (1929) called the “fight or flight” options during an emergency. Whereas fear mobilizes the organism to escape from or avoid an overpowering threat, anger mobilizes the individual to retaliate or resist in the face of provocation or assault, thus opening up another avenue for survival in that situation.

As with many other emotions, the expression of anger also serves a valuable function of social communication (Keltner, Haidt & Shiota, 2006). Even in the absence of aggressive retaliation, a mere display or subvocalization of anger can go a long way to convey power, thereby warding off threats or securing concessions from opponents.

The action tendencies or motivational elements unique to anger do mirror its functions. These tendencies may range from passive resistance to active retaliation (Fernandez, 2008). Thus, the angered individual may be inclined to subvert the attack, to repel it, or to respond with a counterattack. In case damage has already been sustained, the angry person is likely to attempt to redress the wrongdoing by reparation if not retribution. What holds across all these cases is the effort to engage or grapple with the wrongdoer instead of resorting to the escape/avoidance responses that typify fear or the yield/submission responses that typify sadness.

However, it remains an unequivocal position in affect science that anger is essentially a negatively valenced emotion (e.g., Carver & Harmon-Jones, 2009). In other words, the subjective quality of anger is something intrinsically unpleasant. But, certainly, unpleasantness by itself does not warrant intervention. It is when anger poses adverse effects on health and quality of life that it becomes especially problematic. There is now a list of medical conditions linked in some way or the other to anger e.g., the cumulative effect of anger on arteriosclerosis, the precipitation of myocardial infarctions by acute anger (Chapter 25 by J.E. Williams, this book), and the exacerbation of pain by anger (Fernandez & Turk, 1995; Fernandez & Kerns, 2008). Moreover, those prone to anger are more likely to engage in excesses of consumption and self-medication with substances such as narcotics and alcohol. Angry outbursts also potentiate poor judgment leading to risk taking (as in reckless driving) and anger is often implicated in acts of violence and destructiveness (Chapter 17 by J.S. Lerner et al., this book).

When any of the above concomitants reaches such a magnitude that there is impairment in day-to-day functioning, then the anger has become dysfunctional and treatment is indicated. Conversely, treatment is contraindicated if anger does not disturb, impair, or threaten one’s interpersonal or social relationships, occupational life, health, or other areas of functioning. This is congruent with the DSM view of a mental disorder as something that is not just deviant or conflictual but a clinically significant pattern or syndrome of behavior/psychological state that produces distress or disability or poses an increased risk (American Psychiatric Association, 2000). These guideposts do help to discriminate between adaptive and maladaptive anger, and the latter is the target of psychological treatment. The reader is also referred to chapter 27 by R.W. Novaco, this book.

28.1.2 The Target of Anger Treatment

Interchangeable use of the words anger, aggression, and violence can blur the focus of treatment. The three terms are not synonymous. Etymologically, they have undergone variation to convey subtle but meaningful distinctions among naturally occurring phenomena.

In formal definition and also implicit in common parlance, aggression is behavior that is intended to injure or damage (Worchel, Cooper, Goethals, & Olson, 2000). Aggressive acts may emerge out of action tendencies linked to anger. But, it must not be overlooked that the possible motives for aggression are multifarious e.g., thrill (as in pyromania), lust (as may be the case in rape), or greed (as in armed robbery). Anger management is therefore not necessarily aggression management.

As pointed out in chapter 26 by E. Fernandez and A. Wasan, this volume, the injurious consequences of aggression may be bodily harm, psychological hurt, and/or material damage. We define violence as that subtype of aggression in which actual physical harm is inflicted upon an individual. Shouting profanities at the top of one's voice is aggressive but not violent; kicking and punching someone are acts of violence. Violent acts are often attributable to anger, but (like other acts of aggression) they may originate outside the realm of anger – alternatively motivated by greed, lust, thrill, or mere obedience to authority. Strictly speaking, then, violence is not the target of anger management.

Just as aggression and violence can occur without anger, the converse, anger without aggression or violence, is quite possible. Many people experience anger without acting on it or even exhibiting it. (As explained earlier, this does not discount the toll of anger on one's health). To build on what was mentioned earlier, anger is a subjective feeling of unpleasantness bound up with the interpretation of an action as wrongful and tied to an inclination toward defiance or opposition. Anger may result in aggressive behaviors and physical aggression may culminate in physical injury through acts of violence, or else it may not “materialize” into any such behavior. Broadly speaking, the structure of angry feelings is essentially cognitive motivational and this structure must be a prime target of any anger management regimen.

28.2 Past Prescriptions for Anger Management

The literature on anger management contains an assortment of techniques that have been applied in clinical settings or explored in research. Some of these are standalone procedures not tied to any particular school of thinking. Others are grounded in certain philosophical or religious positions. Yet others derive from special psychological theories. It is beyond the scope of this chapter to review the large body of publications in this area. Instead, relevant techniques will receive mention along with a supporting citation and they will be grouped in order to show some important patterns or trends in the psychotherapy for anger.

28.2.1 Standalone Techniques

An example of a standalone procedure is music. Not only musicians but also non-musicians (e.g., Pythagoras) have long extolled the healing power of music. Whether the client's role is active as in a composer or a performer or receptive (that of a listener), there is a potential to profoundly experience and alter emotions (Bunt & Pavlicevic, 2004). Music has been blended with kinesthetic movement (e.g., Meeker, 1985) to alter anger. Percussion has also found utility in the regulation of anger as in Currie (2004). Especially for children, play has been favored as a medium for communicating anger

(e.g., Fischetti, 2001) though this is not to be ruled out for adults either. Without being couched in religion or philosophy, meditation too has been turned into a readily usable technique for anger management (e.g., Dua & Swinden, 1992).

28.2.2 Philosophico-Religious Approaches

The approaches to anger management that are tied to some philosophical/religious position are not merely technical. They are broad perspectives on life. In many ways, these are matters of lifestyle. For example, Stafford (1986) has articulated the Biblical approach to anger management by using numerous parables and teachings from the Bible. Stratton (1923) was one of the first in the west to document eastern approaches to anger management, e.g., Vishnuism and Jainism. Recently, the relevance of Buddhism to anger management has been gaining popular appeal as well as scholarly interest (Bankart, 2006; Levine, 2000). Existential thinking has also provided a frame of reference for anger by transcending what is rational and addressing certain realities of the human condition, e.g., freedom to choose, responsibility, death, and the individual quest for meaning in life (May, 1958). This can provide an attitudinal set that reduces the likelihood of anger (e.g., Augsburger, 1986).

28.2.3 Psychological Approaches

Various movements in psychology have also come to be associated with their own unique brands of anger management. Particularly well known in this regard are anger management approaches based on psychoanalysis, Gestalt therapy, and experiential therapy.

Psychoanalysis as founded on Freudian theory regards (destructive) anger as instinctual, often hidden from the conscious mind, but sometimes manifested in disguise. Modern day object relations theory has built on Freud's ideas by further asserting that early relationships with significant others form the basis of anger in adult life. Therefore, as pointed out by Heinrichs (1987), psychoanalytic therapy for anger is conducted with the expectation that over time, there will be transference of these relationships from the client to the therapist. Certain defense mechanisms like denial, projection, and rationalization of anger may be detected by an astute therapist. The actual alleviation of anger depends much on the client's verbal communication through which insight can be gained into the origins of the anger (Heinrichs, 1986; Knafo & Moscovitz, 2006).

Unlike psychoanalysis, Gestalt therapy is focused on the present rather than distant childhood events. Its founder, Frederick Perls, was fundamentally a phenomenologist, who gave more emphasis to describing rather than explaining in therapy (Perls, 1973). Most important, in the context of anger, is the attention to nonverbal behaviors of the client, because these are regarded as more informative of emotion than are verbalizations. Pointing out such nonverbals is supposed to make the client more aware of his/her anger. The empty chair technique is another hallmark of Gestalt therapy, and its application to anger has been described in an empirical study by Conoley, Conoley, McConnell, and Kimzey (1983). In this procedure, the client talks to an imaginary person in an empty chair so that any previously suppressed anger reaches awareness and can then be safely released. This has the additional benefit of perspective-taking where one sees the other person's point of view while also seeing oneself from the other person's point of view (Day, Howells, Mohr, Schall, & Gerace, 2008). Perls added his own distinctive personal style to Gestalt therapy so that it often appeared blunt and confrontive, though the intent was to evoke emotion, enhance awareness of it, and thereby bring the client closer to a whole (gestalt).

The view that therapy should not be just about talking but about experiencing is captured in a variety of therapies that can be grouped into the experiential school. Psychodrama (Moreno, 1946) is perhaps the best example of this. Really designed for groups, it assigns clients to take on the role of certain characters in a play as if in a theater; it is also improvisational, in which case, much can be revealed about individual emotions such as anger. Modern day examples of experiential therapy go by various terms such as emotion-focused therapy (e.g., Greenberg & Goldman, 2008). What is central to this school is the elevation of emotion to the status of an avenue rather than just an outcome of psychotherapy. Awareness of feelings is encouraged as is the experiencing of these feelings in the clinical context and a changing of the emotional experiences with new experiences. This has been viewed as appropriate for regulating anger (Pica, Engel, & Welches, 2003).

Running counter to the experiential approach is the radical behaviorist view that anger is either a reflex or else an operant (e.g., Clement, 1986). One corollary is that anger-related displays (as in the case of tantrums) can be extinguished by simply ignoring them. Unfortunately, this has variable effectiveness (e.g., Drabman, Jarvie, & Archbold, 1976), not to mention the post tantrum dysphoria that can persist (Potegal & Davidson, 1997). For adults in particular, extinction of the overt behavior may do little to diminish their inner feelings; it may even appear dismissive and lead to aggravation of anger.

A behavioral procedure that has a long history of success in anxiety management and is also applicable to anger control is systematic desensitization. This is premised on the view that anger arousal, like anxiety, can be reciprocally inhibited by relaxation. Therefore, as in the classic study by Rimm, deGroot, Boord, Heiman, and Dillow (1971) a hierarchy of potentially anger-provoking scenarios is constructed in collaboration with the client or the subject. The client is then asked to relax in response to any anger triggered by imagining the least anger-provoking scenario; when the anger arousal has declined as a function of the relaxation response, the next most intense scenario is attempted, and so on, until the individual is systematically desensitized to the whole hierarchy.

Just as a radical behavioral approach to anger reduction is possible, a strict cognitive approach has also been articulated (e.g., Reeder, 1991). As in Beck's (1976) cognitive therapy for depression, this is directed toward the appraisals as well as the elaborate schemas that underlie anger. Any distortions or errors of cognition are corrected through a process of guided discovery that relies heavily on logic and empirical evidence. In addition, new coping statements are introduced into the individual's self-dialogue.

28.2.4 Combinations of Techniques

Certain therapeutic techniques have been combined into anger management packages. In a groundbreaking investigation, Novaco (1975) found value in combining relaxation training plus self-instruction for regulating anger. Since then, this combination of cognitive restructuring and relaxation has been the mainstay of many anger management packages (e.g., Deffenbacher, Story, Stark, Hogg, and Brandon, 1987, 1990). The relaxation may be achieved by progressive muscle relaxation, cue-controlled relaxation, deep breathing, and imagery. The cognitive component (as described in Deffenbacher & Stark, 1992) might entail a reappraisal or a reinterpretation of any overly demanding, overgeneralized, and inflammatory self-dialogue.

Another common package focuses on social skills. Feedback is provided on key behaviors such as listening, eye contact, and vocal tone as well as the basics of dialogue with people with whom one has conflicts. This has been used widely with children (see reviews by Fernandez, 2003b; Morrison & Sandowicz, 1994), adolescents (as reviewed by Fernandez, 2003a; Ollech, 1992), and other special subgroups of youth (e.g., Lochman & Lenhart, 1993). Recently, Day et al. (2006) effectively

used videotaped feedback to enhance the understanding of the sociocultural context of anger among Australian aborigines. An offshoot of social skills training is assertiveness training which is centered around more complex interpersonal communication skills. Within a spirit of frankness and mutual respect, the assertive individual remains calm but firm while stating an opinion, making requests of others, or refusing any unreasonable demands (e.g., Duckworth & Mercer, 2006). Such tact has been embellished with problem solving where there is an attempt to brainstorm for various courses of action, implement the optimal solution, and review the consequences, as in the study by Nezu, Nezu, and Arian (1991).

One cognitive-behavioral package that bears mention is stress inoculation training which was based on theories of cognitive-behavior modification originally applied to anxiety (Meichenbaum, 1975). It was pioneered for anger management by Novaco (1977). Framed in terms of coping skills, stress inoculation comprises three steps: cognitive preparation, skill acquisition, and application training. It is essentially a performance-based intervention during which the client engages in reframing of appraisals, relaxation training, imagery, modeling, and role playing to strengthen his/her ability to cope with anger-provoking situations.

The foregoing literature on anger management is clearly dominated by one hybrid form of therapy: cognitive-behavioral therapy or CBT. This relies primarily on reinforcement techniques to modify behavior and discursive exchanges to alter the way in which information is processed. The latter which is often called reappraisal usually proceeds within the guidelines of rational-emotive therapy (Ellis & Dryden, 1997) so that distorted or erroneous beliefs are disputed on logical and empirical grounds and then replaced by rational alternatives that are supposed to be more adaptive. The relaxation can take the form of a host of techniques of which the most common and convenient are deep breathing and muscle relaxation; the objective, in the context of anger management, is to diminish physical arousal much as in anxiety management where tension and calmness reciprocally inhibit each other.

A meta-analytic synthesis of 50 studies employing such cognitive-behavioral interventions for anger management produced a grand weighted mean effect size of +0.70 (Beck & Fernandez, 1998b). This means that the average treatment subject was better off than 76% of untreated subjects. Inclusion of a few more studies in an updated meta-analytic review of CBT for anger led to an almost identical effect size (DiGuiseppe & Tafrate, 2003). Another quantitative review included several unpublished studies in addition to published studies on CBT for anger and aggressive behavior in children and adolescents (Sukhodolsky, Kassinove, & Gorman, 2004). The mean effect size in this instance was +0.67. Hence, it is evident that CBT is efficacious in the amelioration of anger and there is a strong convergence in the strength of its effect toward +0.70. Interestingly, this is also very close to the classic finding by Smith and Glass (1977) on the overall efficacy of psychotherapy.

28.3 An Integrative Program for Anger Regulation

The growing practice of combining techniques of anger management can be enhanced by more comprehensive integration in keeping with recent developments in psychotherapy. The field of psychotherapy has progressed from its beginning (a century ago) as a monolithic school called psychoanalysis to a period of elaboration in the 1960s and the 1970s when divergent schools of therapy reached the forefront to a new era of integration since the 1990s. To substantiate this point about the advent of this era, there are currently two professional societies dedicated to integrating psychotherapy. One is the Society for the Exploration of Psychotherapy Integration (SEPI) with its official journal, the *Journal of Psychotherapy Integration*. The second is the UK Association

for Psychotherapy Integration (UKAPI), which releases the *British Journal for Psychotherapy Integration*. Responding to growing calls for integration in the practice and study of psychotherapy (e.g., London, 1988; Castonguay & Goldfried, 1994; O'Leary 2006), several reviews and critiques have appeared in journals (e.g., Glass, Arnkoff, & Rodriquez, 1998; Safran & Messer, 1997) in addition to books on theory of psychotherapy integration (e.g., Garfield, 1995; Holmes & Bateman, 2002; Norcross & Goldfried, 2005) and case examples in psychotherapy integration (e.g., Stricker & Gold, 2006).

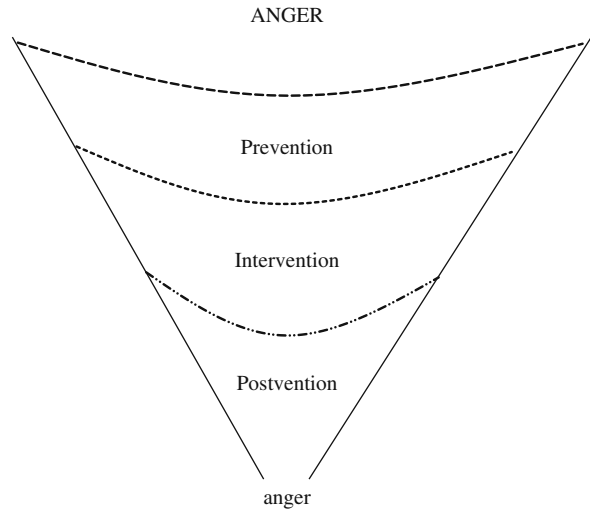
Yet, the field of anger management has been slow to embrace integration. As mentioned earlier, a combination of cognitive and behavioral interventions is evident in the classic work of Novaco and the contributions of Deffenbacher. Much more can be systematically incorporated into anger treatment than the cognitive and behavioral schools; within these schools themselves, more techniques can be harnessed for the regulation of anger.

Part of the rationale for psychotherapeutic integration is the relative lack of any striking differences in the overall efficacy of different schools of psychotherapies. There has also been a recognition that beneath the contrasting jargon of psychotherapy schools may lie some common factors in therapeutic change. Even where differences among schools of thought could not be ironed out, the possibility of strength in diversity has been a lure. It is therefore not surprising that a modal number of psychotherapists (particularly in the United States) now identify themselves as eclectic or integrative (Prochaska & Norcross, 2003).

This opens up the question of how one combines diverse therapeutic strategies – what I call “rules of combination.” At its crudest, the techniques can be combined arbitrarily with the simple expectation that any “cocktail” will surpass its individual ingredients in potency. Another option is what Lazarus (1967) first referred to as technical eclecticism in which techniques are combined so long as they are not incompatible with one another during implementation. A third option goes deeper to select techniques for combination on the basis of theoretical compatibility.

The proposed integrative psychotherapy for anger was developed with regard to technical as well as theoretical compatibility (Fernandez, 1999, 2002). Cognitive strategies are combined with behavior modification techniques because of their common roots in learning theory. Also included is a third school called “affective therapy.” Rooted in experiential therapy, this does not shy away from the feelings or emotions people experience or express. In fact, it can be emotionally evocative; it can also be emotionally cathartic, and new emotional experiences can be formed. The product of this integration across three broad psychotherapeutic paradigms is a “cognitive–behavioral affective therapy” or CBAT for anger (Fernandez, 2002; Herd & Fernandez, 2005). This mirrors the general progress in psychology from behaviorism to cognitivism to the recent resurgence of affect as a worthwhile topic of study. What further distinguishes CBAT from its forerunners is the *sequential* integration of techniques selected (Fernandez, 2001). Some are introduced in a prevention phase, others are delivered in the intervention phase, and the rest are reserved for a final phase of remediation also called “postvention.” The prevention phase consists primarily of behavioral techniques that are somewhat concrete and easy to grasp (e.g., contracting). This is accompanied by the relatively abstract cognitive strategies that dominate the intervention phase; reappraisal, for instance, requires some introspection to identify private mental events plus analytic skills to process them. The final phase comprises affective therapies which deal with what is subjectively felt by the individual; specifically, emotions are regulated by re-experiencing, releasing, or converting them into a safe form. Thus, there is a progression in psychological sophistication or complexity as one moves through the succession of these three phases from the concrete to the abstract to the experiential. The particular sequencing of techniques follows from the dictum that prevention is the sensible starting point in tackling a problem. The prevention phase attempts to forestall the very onset of anger, the intervention phase aims to reduce the intensity or the duration of anger that has already ignited, and

Fig. 28.1 The programmatic treatment of anger by CBAT



the postvention phase is designed to ameliorate or discharge any residual anger. As illustrated in Fig. 28.1, what escapes prevention may later be brought under control through intervention; what defies intervention can be left to the cathartic or palliative procedures of the final affective phase. CBAT thus goes beyond CBT by not only focusing on cognition and behavior as entry points for change but also adding a special route for the modification of affect. After all, anger is an emotion, and to have no room in therapy for the feeling component of this emotion is to deny its very essence. No doubt, special care is needed in implementing these diverse procedures; this point will be touched on later. In sum, CBAT is presented here as a program of carefully selected and sequenced techniques – drawn from cognitive, behavioral, and affective psychotherapies and adapted for the prevention, intervention, and remediation of anger. Each phase attempts to filter out what the preceding phase left behind so that the anger at the end is presumably less than what it was to begin with.

28.3.1 *The Prevention Phase*

Punctuating the start of this phase is psychoeducation during which participants are given a working definition of anger, a rationale for anger regulation that is tied to a description of the ill effects of anger, and a preview of the psychological approach to anger regulation. This is followed by the presentation and signing of a behavioral contract which marks the commencement of active participation in the prevention phase. In the contract is set out the goal of minimizing maladaptive anger and a system of points to be awarded contingent upon different degrees of goal attainment. The points can be totaled at the end of each week for exchange with some appropriate reward or privilege. As is the norm with behavioral contracts, this is not legally binding but in the experience of my research team, participants take it seriously. The contract documents and formalizes their commitment to reduce anger.

What goes hand in hand with the behavioral contract is self-monitoring. This requires participants to self-observe and self-record instances in which they get angry. As in our published studies (Beck & Fernandez, 1998a; Fernandez & Beck, 2001), a chart printed on small pocket size booklets allows participants to graph the intensity of anger as a function of time, from its onset to its peak to its nadir. This allows a computation of the frequency, the intensity, and the duration of anger. The data in turn

can be reviewed in order to determine fulfillment or non-fulfillment of the contract. Moreover, as repeatedly reported in the behavior modification literature, self-monitoring sets in motion a certain reactivity wherein the measured variable begins to change under measurement (Korotitsch & Nelson-Gray, 1999). So, even though it is not regarded as an intervention per se, self-monitoring often ushers in a diminution of the problem behavior.

Behavior rehearsal is another component of the prevention phase. This entails training in the anticipation of anger-provoking events and training in how to respond adaptively to such encounters. The clinician may engage the client in a role play of a characteristic anger-triggering situation. The clinician models the appropriate or desired response and the participant learns to do the same through an iterative process as is commonly the case in social learning.

Response prevention is also employed especially for cases where there is a tendency for escalation. For example, if the participant is inclined to be vociferous and agitated when angry, s/he practices speaking in a lower volume and remaining composed. This is designed to reduce afferent feedback of anger-related physiological arousal. It also holds back anger signals from reaching others who may otherwise respond likewise.

A final component of the prevention phase is stimulus control. In behavior modification nomenclature, a stimulus has discriminative properties such that certain behaviors are more likely to arise in certain contexts. Just as the alcoholic may be admonished to stay away from bars or taverns or even the company of other alcohol abusers, the anger-prone individual may be advised to avoid those situations that so powerfully elicit his/her anger. This form of stimulus control is not meant to be a way of total disengagement; rather, it is meant to remove the client from extreme situations in which there is a certain inevitability of the anger. For instance, a participant in one of our CBAT programs reported that although she had made considerable progress in minimizing her anger in social situations, she remained uncontrollably propelled to anger in her part-time job as a customer service representative for an airline. When it became clear through trial and error that this was virtually intractable and that she had better alternatives, she chose to get out of this chronic situation in favor of an alternative as a medical dictationist. Following that, her anger episodes declined dramatically.

28.3.2 The Intervention Phase

As alluded to earlier, prevention may cast a wide net, but it is only the first in a series of nets. What is preventable in theory may not be forestalled in practice. Should this happen, the next step is to intervene upon the anger that has arisen.

An elementary technique for aborting anger is thought stopping. It entails quietly repeating a word or a phrase that, for the user, connotes the opposite of anger. An example is the word “peace,” or the phrase “settle down.” A neologism can also be adopted as long as (for the individual concerned) it represents something antithetical in meaning to anger. Thought stoppers can serve as an interrupt mechanism early in the onset of anger. After repeated pairing with anger, they may become cue controlled and increase in potency with each instance of effective use.

Should the anger persist, a more elaborate strategy is called for: reappraisal. Basically, the angered person’s attributions are identified, critically evaluated, and replaced by more rational alternatives. One of the foci of reappraisal is the perceived damage – something which upon re-evaluation often turns out to be less than initially assumed. Even if the consequences are dire, the question of intentionality on the part of the offending person must be considered. Experiments in affect science have consistently shown that an appraisal that the offender intended the offense is a pivotal requirement for anger (e.g., Ben-Zur & Breznitz, 1991; Yamaguchi, 1996). If the subject can learn to question his/her usual automatic appraisal that an offense was intended, then a reduction

in anger is more attainable. There can be several other issues for reappraisal, including the possible role of the angered person; if self-culpability is acknowledged, anger is likely to diminish further.

If anger does not respond to these elaborate reappraisals, it may then be subjected to distraction. Ranging from passive options such as watching television to active options such as going for a walk, these strategies share in common the objective of diverting attention from the anger-provoking situation. Again, this is adaptive because when one cannot change one's mind about something troubling, it is better to take one's mind off it – at least for the short term.

Relaxation is an imperative tool in the repertoire for regulating anger-related arousal. Consistent with the cognitive emphasis in the intervention phase, autogenic relaxation is prescribed. Here, the task is to self-suggest relaxation in various sites and systems of the body. For example, the individual may imagine a sensation of coolness spreading across the body, a release of tension from various tight muscle groups, and a slowing down of heart rate and blood pressure (i.e., opposing the physiological changes associated with anger; Chapter 7 by G. Stemmler, this book). By efferent pathways, such suggestions have been known to facilitate actual reduction in physiological arousal (Stemmler, Aue & Wacker, 2007). The salutary effects of autogenic relaxation may also be enhanced by situational imagery, e.g., images of natural beauty which are generally regarded as inconducive to anger. There is no reason to exclude other techniques of relaxation too. Thus, autogenic relaxation may be supplemented as needed with diaphragmatic breathing and Jacobsonian muscle relaxation (McCallie, Blum, & Hood, 2006). The objective remains the same: to reduce the arousal that often compounds the experience and expression of anger.

When anger has been curbed or abolished by the above interventions, the success may be accompanied by self-statements, e.g., “I did it,” “I triumphed over anger,” “I deserve to feel good.” This would serve to reinforce the future use of effective techniques and it would also strengthen the individual's sense of self-control over anger.

28.3.3 *The Postvention Phase*

As with prevention, intervention is not a guarantee of cure. Some angry feelings may persist even after procedures like reappraisal, distraction, and relaxation. Should that happen, they can be taken up in one last phase: postvention. This involves regulating the subjective feelings of anger using select techniques from the affective therapies.

One option is the empty chair technique from Gestalt therapy. Here, the angered individual takes a seat opposite another empty seat in which is the imagined offender. Then begins the verbal expression of anger in the *here-and-now*. This is particularly productive if the grievance has stayed unresolved due to inaccessibility of the offending person (perhaps because of death, distance, or other factors). The exercise facilitates a release of pent-up emotion. It is not catharsis in the sense of anger being stirred up, ruminated upon, or vented in some primal way such as punching a bag; such procedures can indeed leave a person feeling more anger (e.g., Bushman, 2002). Rather, the Gestalt empty chair technique allows the aggrieved person to safely re-experience and express feelings that have been withheld. It is in such instances that the empty chair technique has been found to significantly reduce anger (Conoley et al., 1983).

For those who prefer writing to talking, a letter may be composed about the unresolved anger. The individual is given the license to write down his/her feelings in all their richness and complexity, as in the work of Pennebaker and Seagal (1999) and Pennebaker and Stone (2004). Factual details are also admissible, though the emphasis is on emotion and related action tendencies. In a classic book,

White and Epston (1989) have described several variations of narrative writing and correspondence that serve as avenues for therapeutic confession and expression of emotions like anger. For instance, once written, a letter may be disposed of as a sign of closure; alternatively, the letter may be read by a therapist *qua* confidante before the final ritual of disposal. Even though quantitative data on efficacy are scant, anecdotal support for such techniques has been provided by eminent scholars and clinicians such as Mahoney (1991).

Where language is limited for one reason or another, art offers a universally applicable medium for the expression of anger. Painting, for instance, has been and can be used to represent angry feelings that are sometimes difficult to articulate. Sculpture, sketching, and musical performance offer yet other options to suit a variety of individual preferences. While quantitative outcome data for such procedures is lacking, this remains a common clinical practice supported by numerous anecdotal accounts of its therapeutic value (e.g., Liebmann, 2008; Malchiodi, 2005).

In a grand finale, conclusion of the CBAT program is marked by a ceremony in which each participant is congratulated with a certificate of completion. What the contract is to the start of treatment, the completion certificate is to the close of treatment. Having completed the CBAT program, the participant should have had adequate opportunities to reduce his/her maladaptive anger and acquire a set of skills for application to naturalistic situations. But it does not signal the end of the endeavor. Completers are encouraged to keep practicing the skills to prevent, intervene upon, or remediate maladaptive anger in their world beyond the clinic. Ideally, booster sessions of training are imperative as are follow-up sessions for ascertaining the generalization of treatment gains beyond the clinical context.

28.3.4 CBAT Outcome

Some preliminary findings are available on the evaluation of CBAT for anger. Fernandez and Scott (2009) reported on the outcome of a minimal four-session program of CBAT implemented in a sample of chemically dependent individuals referred for anger management. Results from a sample of 26 (equally divided between males and females) revealed significant changes in the desired direction on each of the six subscales of the State Trait Anger Expression Inventory (Spielberger, 1988) between pre-treatment, post-treatment, and one-month follow-up. Self-monitored frequency of anger also declined significantly across all these phases. The pre-post effect size was highest in the case of trait anger, $d = +0.99$; for state anger, $d = +0.80$.

In a separate study of self-referred individuals from a community sample, Herd and Fernandez (2005) reported on a CBAT group compared to a minimalist treatment group that engaged in self-monitoring. Dependent measures included self-monitored measures of anger in naturalistic settings. The results showed significant reductions in the CBAT group but not its counterpart. Furthermore, a between-groups effect size of +0.53 was obtained in favor of CBAT. In an extension of this design, the CBAT group was compared with an expanded CBT group that received identical cognitive-behavioral techniques for prevention and intervention but no affective techniques. In both cases, the program consisted of the bare minimum of 4 weeks plus follow-up. In addition to self-monitored frequency, duration, and intensity of anger, standardized test measures were used as dependent variables. Results at this stage have revealed significant reductions in nearly all dependent measures for anger in both groups. However, the average pre-post effect size for CBAT was in the vicinity of +1.0, which was higher than that for CBT. This points to the value of an integrative approach to anger regulation in which affective techniques are incorporated in addition to a wide variety of cognitive and behavioral techniques.

28.4 Further Considerations

It is beyond the charge of the present chapter to discuss the myriad issues pertaining to CBAT implementation and outcome. These include the nuances of screening, the choice of assessment instruments, ensuring adherence, the duration and dosage of treatment, the transfer of gains from the clinic to the natural environment, the challenges of individual versus group formats of treatment, and the tailoring of treatments to developmental and demographic variables. Such issues are common to most therapeutic regimens. In general, support has emerged for the efficacy of the CBAT program in regulating anger of college students, substance abuse patients, and a community sample of volunteers with a history of maladaptive anger (Fernandez & Beck, 2001; Fernandez & Scott, 2009; Herd & Fernandez, 2005). A treatment manual is currently in preparation to provide guidelines for implementation of CBAT in diverse populations. This is supplemented with a methodology for evaluation of the outcome of this program in the prevention, intervention, and postvention of anger.

References

- American Psychiatric Association (2000). *Diagnostic and Statistical Manual of Mental Disorders Fourth Edition Text Revision (DSM-IV-TR)*. Washington, DC: American Psychiatric Publishing.
- Augsburger, D. (1986). An existential approach to anger management training. *Journal of Psychology and Christianity*, 5, 25–29.
- Bankart, C. P. (2006). Treating anger with wisdom and compassion: A Buddhist approach. In E. L. Feindler (Ed.), *Anger related disorders: A practitioner's guide to comparative treatments* (pp. 231–255). New York: Springer.
- Beck, A. T. (1976). *Cognitive therapy and the emotional disorders*. New York: International Universities.
- Beck, R. & Fernandez, E. (1998a). Cognitive-behavioral self-regulation of the frequency, duration, and intensity of anger. *Journal of Psychopathology and Behavioral Assessment*, 20, 217–229.
- Beck, R. & Fernandez, E. (1998b). Cognitive-Behavioral Therapy in the treatment of anger: A meta-analysis. *Cognitive Therapy and Research*, 22, 63–74.
- Ben-Zur, H. & Breznitz, S. (1991). What makes people angry: Dimensions of anger evoking events. *Journal of Research in Personality*, 25, 1–22.
- Bunt, L. & Pavlicevic, M. (2004). Music and emotion: Perspectives from music therapy. In P. N. Juslin & J. A. Sloboda (Eds.), *Music and emotion: Theory and research*. New York: Oxford University Press.
- Bushman, B. J. (2002). Does venting anger feed or extinguish the flame? Catharsis, rumination, distraction, anger and aggressive responding. *Personality and Social Psychology Bulletin*, 28, 724–731.
- Cannon, W. B. (1929). *Bodily changes in pain, hunger, fear, and rage* (2nd ed.). New York: Appleton.
- Carver, C. S. & Harmon-Jones, E. (2009). Anger is an approach-related affect: Evidence and implications. *Psychological Bulletin*, 135, 183–204.
- Castonguay, L. G. & Goldfried, M. R. (1994). Psychotherapy integration: An idea whose time has come. *Applied and Preventive Psychology*, 3, 159–172.
- Clement, P. W. (1986). Behavioral approaches to anger management training. *Journal of Psychology and Christianity*, 5, 41–49.
- Conoley, C., Conoley, J., McConnell, J. A., & Kimzey, C. E. (1983). The effect of the ABC's of rational emotive therapy and the empty-chair technique of Gestalt therapy on anger reduction. *Psychotherapy: Theory, Research and Practice*, 20, 112–117.
- Currie, M. (2004). Doing anger differently: A group percussion therapy for angry adolescent boys. *International Journal of Group Psychotherapy*, 54, 275–294.
- Day, A., Howells, K., Mohr, P., Schall, E., & Gerace, A. (2008). The development of CBT programmes for anger: The role of interventions to promote perspective-taking skills. *Behavioural and Cognitive Psychotherapy*, 36, 299–312.
- Day, A., Howells, K., Nakata, M., Davey, L., Wanganeen, R., & De Santolo, J. (2006). The development of culturally appropriate anger management programs for indigenous people in Australian prison settings. *International Journal of Offender Rehabilitation and Comparative Criminology*, 50, 520–539.
- Deffenbacher, J. L., McNamara, K., Stark, R. S., & Sabadell, P. M. (1990). A combination of cognitive, relaxation, and behavioral coping skills in the reduction of general anger. *Journal of College Student Development*, 31, 351–358.

- Deffenbacher, J. L. & Stark, R. S. (1992). Relaxation and cognitive-relaxation treatments of general anger. *Journal of Counseling Psychology*, 39, 158–167.
- Deffenbacher, J. L., Story, D. A., Stark, R. S., Hogg, J. A., & Brandon, A. D. (1987). Cognitive-relaxation and social skills interventions in the treatment of anger. *Journal of Counseling Psychology*, 34, 171–176.
- DiGuiseppe, R. & Tafrate, R. C. (2003). Anger treatment for adults: A meta-analytic review. *Clinical Psychology: Science and Practice*, 10, 70–84.
- Drabman, R. S., Jarvie, G. J., & Archbold, J. (1976). The use and misuse of extinction in classroom behavioral programs. *Psychology in the Schools*, 13, 470–476.
- Dua, J. K. & Swinden, M. L. (1992). Effectiveness of negative-thought-reduction, meditation, and placebo training treatment in reducing anger. *Scandinavian Journal of Psychology*, 33, 135–146.
- Duckworth, M. P. & Mercer, V. (2006). Assertiveness training. In J. E. Fisher & W. T. O'Donohue (Eds.), *Practitioner's guide to evidence-based psychotherapy* (pp. 80–92). New York, NY: Springer Science + Business Media.
- Ellis, A. & Dryden, W. (1997). *The practice of rational emotive therapy* (2nd ed.). New York: Springer.
- Fernandez, E. (1999). *Integrating therapeutic techniques: The case of anger*. The First Mid-Atlantic Conference, Society for Psychotherapy Research, Baltimore.
- Fernandez, E. (2001). *Sequential integration of cognitive-behavioral and experiential techniques for regulating anger*. Workshop conducted at the Annual Conference of the Society for the Exploration of Psychotherapy Integration, Santiago, Chile.
- Fernandez, E. (2002). *Anxiety, depression, and anger in pain: Research findings and clinical options*. Dallas, TX: Advanced Psychological Resources.
- Fernandez, E. (2003a). Anger regulation in adolescence. In T. Gullotta & M. Bloom (Eds.), *Encyclopedia of primary prevention and health promotion* (pp. 195–199). New York: Kluwer Academic/Plenum Publishers.
- Fernandez, E. (2003b). Anger regulation in childhood. In T. Gullotta & M. Bloom (Eds.), *Encyclopedia of Primary Prevention and Health Promotion* (pp. 190–195). New York: Kluwer Academic/Plenum Publishers.
- Fernandez, E. (2008). The angry personality: A representation on six dimensions of anger expression. In G. J. Boyle, D. Matthews & D. Saklofske (Eds.), *International handbook of personality theory and testing: Vol. 1: Personality theories and models* (pp. 402–419). London: Sage.
- Fernandez, E. & Beck, R. (2001). Cognitive-behavioral self-intervention versus self-monitoring of anger: Effects on anger frequency, duration, and intensity. *Behavioural and Cognitive Psychotherapy*, 29, 345–356.
- Fernandez, E. & Kerns, R. D. (2008). Anxiety, depression, and anger: The core of negative affect in medical populations. In G. J. Boyle, D. Matthews & D. Saklofske (Eds.), *International Handbook of Personality Theory and Testing: Vol. 1: Personality Theories and Models* (pp. 659–676). London: Sage.
- Fernandez, E. & Scott, S. (2009). Anger treatment in chemically-dependent inpatients: Evaluation of phase effects and gender. *Behavioural and Cognitive Psychotherapy*, 37, 431–447.
- Fernandez, E. & Turk, D. C. (1995). The scope and significance of anger in the experience of chronic pain. *Pain*, 61, 165–175.
- Fischetti, B. A. (2001). Use of play therapy for anger management in the school setting. In Drewes, A. A., Carey, L. J., & Schaefer, C. E. (Eds.), *School-based play therapy* (pp. 238–255). Hoboken, NJ: Wiley.
- Garfield, S. L. (1995). *Psychotherapy: An eclectic-integrative approach*. New York: Wiley.
- Glass, C. R., Arnkoff, D. B., & Rodriguez, B. F. (1998). An overview of directions in psychotherapy integration research. *Journal of Psychotherapy Integration*, 8, 187–209.
- Greenberg, L. S. & Goldman, R. N. (2008). *Emotion-focused couples therapy: The dynamics of emotion, love, and power* (pp. 351–364). Washington, DC, USA: American Psychological Association.
- Heinrichs, D. J. (1987). A psychoanalytic approach to anger management training. *Journal of Psychology and Christianity*, 5, 12–24.
- Herd, V. & Fernandez, E. (2005). *The Efficacy of Integrative CBAT in regulating maladaptive anger*. The 21st Annual Meeting of the Society for the Integration of Psychotherapy. Toronto, Canada.
- Holmes, J., & Bateman, A. (Eds.). (2002). *Integration in psychotherapy: Models and methods*. New York: Oxford University Press.
- Keltner, D., Haidt, J. & Shiota, M.N. (2006). Social functionalism and the evolution of emotions. In M. Schaller, J.A. Simpson & D.T. Kenrick (Eds.), *Evolution and social psychology* (pp. 115–142). Madison, CT: Psychosocial Press.
- Knafo, D. & Moscovitz, S. (2006). Psychoanalytic treatment of anger and aggression. In E. L. Feindler (Ed.), *Anger related disorders: A practitioner's guide to comparative treatments* (pp. 97–114). New York: Springer.
- Korotitsch, W. J. & Nelson-Gray, R. O. (1999). An overview of self-monitoring research in assessment and treatment. *Psychological Assessment*, 11, 415–425.

- Lazarus, A. A. (1967). In support of technical eclecticism. *Psychological Reports*, 21, 415–416.
- Levine, M. (2000). *The positive psychology of Buddhism and Yoga: Paths to a mature happiness: With a special application to handling anger*. Mahwah, NJ: Lawrence Erlbaum.
- Liebmann, M. (2008). *Art therapy and anger*. London, England: Jessica Kingsley Publishers.
- Lochman, J. E. & Lenhart, L. A. (1993). Anger coping intervention for aggressive children: conceptual models and outcome effects. *Clinical Psychology Review*, 13, 785–805.
- London, P. (1988). Metamorphosis in psychotherapy: Slouching toward integration. *Journal of Integrative and Eclectic Psychotherapy*, 7, 3–12.
- Mahoney, M. J. (1991). *Human change processes: The scientific foundations of psychotherapy*. New York: Basic Books.
- Malchiodi, C. A. (2005). Art therapy. In C. A. Malchiodi (Eds.), *Expressive therapies* (pp. 16–45). New York, NY, US: Guilford Press.
- May, R. (1958). Contributions of existential psychotherapy. In R. May, E. Angel & H. E. Ellenberger (Eds.), *Existence: A new dimension in psychiatry and psychology* (pp. 37–92). New York: Basic Books.
- McCallie, M. S., Blum, C. M., & Hood, C. J. (2006). Progressive Muscle Relaxation. *Journal of Human Behavior in the Social Environment*, 13, 51–66.
- Meeker, A. M. (1985). “Empty Lizzie”: Using the expressive arts as a tool for dealing with anger. *The Pointer*, 29, 31–34.
- Meichenbaum, D. H. (1975). *Stress inoculation training*. New York: Pergamon.
- Moreno, J. L. (1946). *Psychodrama* (2nd ed, Vol. 1). New York: Beacon House.
- Morrison, G. M. & Sandowicz, M. (1994). Importance of social skills in the prevention and intervention of anger and aggression. In M. J. Furlong, D. C. Smith (Eds.), *Anger, hostility, and aggression: Assessment, prevention, and intervention strategies for youth* (pp. 345–392). Brandon, VT: Clinical Psychology Publishing Co.
- Nezu, C. M., Nezu, A. M., & Arean, P. (1991). Assertiveness and problem-solving training for mildly mentally retarded persons with dual diagnoses. *Research in Developmental Disabilities*, 12, 371–386.
- Norcross, J. C., & Goldfried, M. R. (Eds.). (2005). *Handbook of psychotherapy integration* (2nd ed). New York: Oxford University Press.
- Novaco, R. W. (1975). *Anger control: The development and evaluation of an experimental treatment*. Lexington, MA: D.C. Heath.
- Novaco, R. W. (1977). Stress inoculation: A cognitive therapy for anger and its application to a case of depression. *Journal of Consulting and Clinical Psychology*, 45, 600–608.
- Ollech, D. (1992). *Adolescent assertiveness and social skills training: A clinical handbook* (pp. 151–164). New York: Springer.
- O’Leary, E. (2006). The need for integration. In E. O’Leary & M. Murphy (Eds.), *New approaches to integration in psychotherapy* (pp. 3–11). New York: Routledge/Taylor & Francis Group.
- Pennebaker, J. W. & Seagal, J. D. (1999). Forming a story: The health benefits of narrative. *Journal of Clinical Psychology*, 55, 1243–1254.
- Pennebaker, J. W. & Stone, L. (2004). Translating traumatic experiences into language: Implications for child abuse and long-term health. In L. J. Koenig, L. S. Doll, A. O’Leary & W. Pequegnat (Eds.), *From child sexual abuse to adult sexual risk: Trauma, revictimization, and intervention* (pp. 201–216). Washington, DC: American Psychological Association.
- Perls, F. S. (1973). *The Gestalt approach*. Palo Alto, CA: Science and Behavior Books.
- Pica, M., Engel, S. D., & Welches, P. (2003). An experiential approach to the inpatient anger management group. *International Journal of Group Psychotherapy*, 53, 177–200.
- Potegal, M. & Davidson, R. J. (1997). Young children’s post-tantrum affiliation with their parents. *Aggressive Behavior. Special Issue: Appeasement and reconciliation*, 23, 329–341.
- Prochaska, J. O. & Norcross, J. C. (2003). *Systems of psychotherapy: A transtheoretical analysis* (5th ed.). Belmont, CA: Wadworth.
- Reeder, D. M. (1991). Cognitive therapy of anger management: Theoretical and practical considerations. *Archives of Psychiatric Nursing*, 3, 147–150.
- Rimm, D. C., deGroot, J. C., Boord, P., Heiman, J., & Dillow, P. V. (1971). Systematic desensitization of an anger response. *Behaviour Research and Therapy*, 9, 273–280.
- Safran, J. D. & Messer, S. B. (1997). Psychotherapy integration: A postmodern critique. *Clinical Psychology: Science and Practice*, 4, 140–152.
- Smith, M. L. & Glass, G. V. (1977). Meta-analysis of psychotherapy outcome studies. *American Psychologist*, 32, 752–760.
- Spielberger, C. D. (1988). *Manual for the state-trait anger expression inventory (STAXI)*. Odessa, FL: Psychological Assessment Resources, Inc. (PAR).

- Stafford, C. H. (1986). A biblical approach to anger management training. *Journal of Psychology and Christianity*, 5, 5–11.
- Stemmler, G., Aue, T., & Wacker, J. (2007). Anger and fear: Separable effects of emotion and motivational direction on somatovisceral responses. *International Journal of Psychophysiology*, 66, 141–153.
- Stratton, G. M. (1923). The unangry religions: Taoism, Vishnuism, Buddhism, Jainism. In G. M. Stratton (Ed.), *Anger: Its religious and moral significance* (pp. 96–118). New York, NY, US: MacMillan.
- Stricker, G., & Gold, J. (Eds.). (2006). *A casebook of psychotherapy integration*. Washington, DC: American Psychological Association.
- Sukhodolsky, D. G., Kassinove, H., & Gorman, B. S. (2004). Cognitive-behavioral therapy for anger in children and adolescents: meta-analysis. *Aggression and Violent Behavior*, 9, 247–269.
- White, M. & Epston, D. (1989). *Literate means to therapeutic ends*. Adelaide, SA, Australia: Dulwich Centre Publications.
- Worchel, S., Cooper, J., Goethals, G.R., & Olson, J.M. (2000). *Social psychology*. Belmont, CA: Wadsworth/Thomson Learning.
- Yamaguchi, H. (1996). The expression of anger and aggression in daily life. *Japanese Journal of Experimental Social Psychology*, 36, 273–286.

Part IX
**Conflict and Anger in Family, Workplace,
and Community**

Chapter 29

The Contribution of Child Anger and Fear, and Parental Discipline to Early Antisocial Behavior: An Integrative Model

James Snyder, Lynn Schrepferman, Amber McEachern, and Jamie DeLeeuw

Abstract This chapter examines how parental discipline, child propensities to displays of anger and fear, and child frontal executive inhibition and verbal skills additively and synergistically contribute to overt (aggressive) and covert (sneaky) forms of antisocial behavior. Overt and covert forms of antisocial behavior emerge on different developmental timetables and result from both common and unique sets of variables and variable combinations. Covert antisocial behaviors such as stealing and lying are associated with harsh and angry parental discipline, good child verbal skills and their interaction. Overt antisocial behaviors such as aggression and defiance are associated with parental nattering and inconsistent discipline, especially in the context of poor child frontal inhibition, and low child fear. The chapter exemplifies models which describe development in ways that integrate children's socialization, emotion expression, and capacity for behavior and emotion regulation.

29.1 Introduction

Significant advances have been made in the study of emotions (Lewis & Haviland-Jones, 2000), in affective neuroscience (Derryberry & Tucker, 2006) and in research on socialization (Bugental & Grusec, 2006; Rubin, Bukowski, & Parker, 2006). Each of these areas is relevant to understanding the development of both adaptive and maladaptive behaviors. However, research in each area has often been pursued without clear linkages to the other areas. This chapter attempts to bridge these areas to build and test a multi-process, social developmental model of how anger contributes to risk of early antisocial behavior.

The proposed integrated model is built on the following premises. Anger and other basic emotions are adaptive bio-behavioral responses built into humans to meet organismic needs and environmental demands. Successful adaptation entails the ability to engender, utilize and regulate the full range of basic emotions in a situation-specific, balanced, and flexible manner to simultaneously promote individual well-being and sustain supportive social relationships (Izard, 2002). Basic emotions are neither “good” nor “bad” per se. Rather, they are potentially functional, motivational processes which instigate and coordinate cognitive, neural, and physiological activity in the service of goal-directed behavior (Ekman & Davidson, 1994).

J. Snyder (✉)
Wichita State University, Wichita, KS, USA
e-mail: james.snyder@wichita.edu

The experience, regulation, and expression of emotions involve multiple processes. Salient unconditioned and conditioned positive and aversive environmental events trigger activation of neural networks in the limbic system associated with strong emotional arousal. These limbic networks organize and instigate physiological arousal, facial and vocal displays, and approach and avoidance behaviors. Activity in these limbic networks also engages frontal executive control processes which may then regulate emotional arousal and its behavioral expression based on past experience, current environmental cues and contingencies, and long-term goals (Luciana, 2006).

Goal-directed behavior is shaped and maintained by social environmental events as well as by intra-individual emotional–motivational and executive control regulatory processes. However, a simple additive model combining emotional arousal, executive control processes, and social environmental experiences is inadequate. Emotions and executive regulatory processes are evoked and shaped by environmental events and, in turn, affect the likelihood that specific environmental events will be encountered and how those events will be experienced. From this perspective, a full understanding of the contribution of anger to adaptive or problem behavior may require examination of the collective and synergistic influence of emotional, executive control, and social environmental processes. A simplified application of the integrated multi-process model to the development of antisocial behavior is shown in Fig. 29.1.

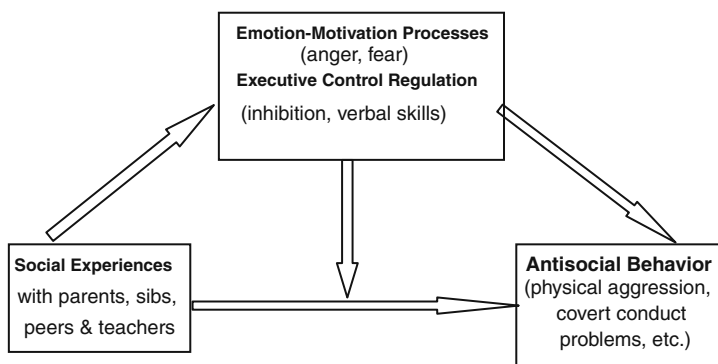


Fig. 29.1 Contributions of emotion–motivational processes, executive control regulation, and social experience to the development of antisocial behavior

The proposed multi-process model is elaborated in three ways in this chapter. First, the various components and hypothesized relationships comprising the model are described, using the role of anger in the development of antisocial behavior as an exemplar. Second, the model is tested using data from the School Transitions longitudinal study of the development of child antisocial behavior. Third, the results are used to examine the utility of the model in relation to further research on the contribution of anger and other emotions to adaptive and maladaptive behaviors.

29.2 Development of Antisocial Behavior

Richard Tremblay (2003) has made a strong case that research on disruptive and norm-violating behavior has been hampered by problems in taxonomy. Behavior that violates societal rules or evokes distress in others has been variously labeled as externalizing, oppositional, disruptive, conduct disordered, delinquent, aggressive, criminal, and antisocial. These labels combine narrow behavior

classes in various ways, and each of these classes may have unique as well as shared developmental trajectories, origins, and sequelae. In this chapter, the term antisocial (AS) is used to refer to the broad, generic class of norm-violating behaviors. Tremblay argues research on the etiology and treatment of AS behavior might be better served by focusing on relatively narrow topographical or functional problem behavior classes, such as physical aggression, verbal and relational aggression, and covert problem behaviors such as stealing and lying. However, the developmental trajectories of each of these narrower classes of AS behaviors are concurrently and sequentially inter-related so that research on each class needs to be integrated with that of other classes. The focus here will be on the role of anger and fear in the origins and development of two relatively narrow, functionally distinct behavior problem classes: physical aggression (P-AGG) and covert problem behaviors (COV).

Empirical support for the distinction between overt AS behaviors (such as P-AGG) and COV such as lying and stealing has been provided by factor analyses of global reports of child behavior problems by parents and teachers (Frick et al., 1993) and by multi-method (Hinshaw, Simmel, & Heller, 1995) and observational data (Willoughby, Kupersmidt, & Bryant, 2001). These analyses indicate that AS behavior can be better represented by two factors rather than just one combined factor. An alternate, more typical perspective combines P-AGG and COV problem behaviors into a broader class labeled conduct problems or antisocial behavior (Dishion & Patterson, 2006). Concurrent measures of overt P-AGG and COV factors are, in fact, significantly correlated (r 's from 0.50 to 0.70), but mono-informant halo effects likely inflate estimates of shared variance.

Longitudinal data indicate that the onset, the persistence, and the growth of P-AGG and COV are different. P-AGG normatively appears as early as 12 months and increases in rate until about 4 years of age after which it decreases (Tremblay, 2003). Sizeable individual differences in P-AGG are observable as early as age 18 months. Individual differences in the amount, direction, and timing of developmental changes in P-AGG are apparent throughout childhood and adolescence (Broidy et al., 2003). About 5% of boys and a smaller percentage of girls display high rates of P-AGG that persist into adolescence. Another 25% of boys and 10% of girls show delayed declines in P-AGG through ages 6–10 years. The remaining majority of children show earlier, normative developmental declines in rates of P-AGG, beginning by age 4 and well under way by age 6 years (Broidy et al., 2003).

The overall trend is for COV to emerge by age 4 years, developmentally later than P-AGG. Rates of COV increase thereafter, with a notable acceleration during early to middle adolescence (Loeber & Stouthamer-Loeber, 1998). Individual differences in trajectories of COV are apparent by age 4 and continue to age 18 (Bongers, Koot, van der Ende, & Verhulst, 2003). The 1- to 2-year stability correlations for COV are modest and increase with age (from 0.20 at 4 years to 0.44 at 8 years), whereas those for P-AGG are larger and change minimally with age (from 0.63 at 4 years to 0.68 at 8 years; Verhulst, Koot & Berden, 1990). The severity of P-AGG and COV increases with their persistence. For example, hitting escalates to assault; minor lying and stealing to burglary and theft; and minor property damage to vandalism and fire setting (Loeber & Stouthamer-Loeber, 1998).

As shown in Fig. 29.2, there are several ways in which the varying developmental trajectories for P-AGG and COV may combine during early and middle childhood to contribute to the overall trends noted above (Loeber, Keenan, & Zang, 1997; Patterson, 1993). For example, COV may progressively replace P-AGG in middle childhood and adolescence so that children who engage in high-frequency P-AGG earlier in development show diminishing P-AGG as P-AGG is increasingly replaced by COV (top left panel in Fig. 29.2). Alternatively COV may overlay earlier P-AGG during middle childhood so that children display high levels of both P-AGG and COV (Patterson, 1993; top right panel of Fig. 29.2). Finally, there may be some children who do not add COV onto earlier P-AGG and consequently evidence persisting P-AGG with low-frequency COV in middle childhood (bottom left panel in Fig. 29.2). There is little evidence of a trajectory for COV during middle childhood

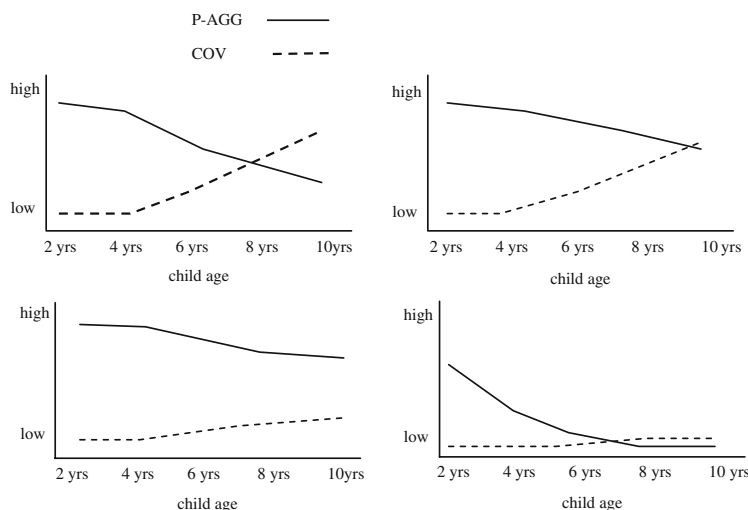


Fig. 29.2 Trajectory classes for the development of physical aggression and covert conduct problems in middle childhood

without earlier P-AGG. From a cross-sectional perspective, children with AS behavior problems during middle childhood might be classified as displaying predominantly P-AGG, predominantly COV, or a combination of P-AGG and COV (Patterson, 1982). Of course, there are a large number of children who evidence normative declines in P-AGG and who do not initiate COV during middle childhood (bottom right panel in Fig. 29.2).

These variations in P-AGG and COV development raise several salient questions. Why is the normative decline in P-AGG delayed for some individuals? Why does COV have a later onset than does P-AGG and increase rather than decline with age? Why do some children show early onset and growth of COV, whereas other children primarily display COV later in development? How can trajectories for P-AGG and COV evidence different age dynamics but still be inter-related? A model integrating child anger and fear reactivity, executive control processes, and family socialization may provide empirical leverage in answering these questions.

29.3 Emotional–Motivational Processes and Frontal Executive Regulation

The conceptual approaches to emotional–motivational processes and frontal executive regulatory systems used in this chapter are derived from differential emotions theory (DET; Izard, 1991) and affective neuroscience (Derryberry & Tucker, 2006). DET suggests the experience and display of anger and other primary emotions (only fear in addition to anger will be considered here) serve different motivational functions. Anger mobilizes energy, activates vigorous approach behavior, and fosters persistence in the face of threat and obstacles to reward (see, e.g., Chapter 21 and also Chapter 17). Fear motivates safety seeking in the face of dangerous environmental events. Anger and fear are adaptive responses to salient environmental opportunities and challenges, and work together as complementary motivational systems (Izard, 1991). Risk for maladjustment increases as there is imbalance in utilization or a failure to effectively regulate either or both emotions as reflected in frequency, duration, or intensity of their expression. Relative imbalance in utilization and dysregulation of anger and fear may contribute to risk for AS behavior, and the exact nature of their imbalance or dysregulation may differentiate risk for P-AGG and COV.

Affective neuroscience (Gray, 1994; Panksepp, 1998; Rothbart, Posner, & Kieras 2006) suggests that two limbic emotional–motivation networks are likely to be activated in response to the environmental occurrence of aversive (punishing) stimuli or frustrative non-reward. One network facilitates anger, behavioral activation, and attack. The second facilitates fear, behavioral inhibition, freezing (waiting), and withdrawal. These two networks serve complementary functions. Each is needed to respond to salient environmental events, and activation of one system tends to inhibit the other.

Activation and balance between limbic networks associated with anger and fear are subject to regulation from executive control networks located in the frontal cortex of the brain, especially the dorsal and the ventral anterior cingulate cortex (Luciana, 2006; Posner & Rothbart, 2007). These executive control networks regulate emotional arousal, inhibit behavior, foster careful monitoring of environmental cues and contingencies, and instigate effortful cognitive appraisal and problem solving. Complete models of the roles of anger and fear in the development of P-AGG and COV should incorporate influence resulting from executive control regulation.

A number of studies support the notion that AS behavior is associated with high impulsivity (or inadequate executive control) and frequent anger, and with low verbal skills, physiological arousal, and fear (Lahey & Waldman, 2003; Moffitt, Lynam & Silva, 1994). But risk for P-AGG and COV may be generated by distinct combinations of these individual characteristics. Emotional reactivity, executive control, and verbal skills also change as a result of maturation and experience (Posner & Rothbart, 2007). We specify how these developmental changes may contribute to age differences in the appearance and growth of P-AGG and COV in a later section.

29.4 Social Learning in Child–Parent Interaction

Several decades of observational research indicates that children's oppositional behavior and P-AGG are shaped by an accumulation of aversive, non-contingent, and unsupportive daily social experiences with parents, siblings, and peers. These aversive events entail both punishment and frustrative non-reward (Patterson, 1982; Patterson, Reid & Dishion, 1992; Snyder, 2002; Snyder & Stoolmiller, 2002). Frequent exposure to aversive social events results in bursts of reciprocal aversive exchange during social interaction (Snyder, Stoolmiller, Wilson, & Yamamoto, 2003; Stoolmiller, 1992) that, fueled by anger, escalate from the verbal and vocal (protests, threats, and screaming) to the physical (P-AGG: kicking, hitting, and biting; Snyder, Edwards, McGraw, Kilgore, & Holton, 1994). Opposition, verbal distress, and P-AGG are further shaped and sustained by short-term social contingencies. The frequency with which oppositional behavior, verbal aggression, and P-AGG are performed is closely tied to their functional value in deflecting aversive events and control by others (negative reinforcement) and in overcoming impediments to accessing expected rewards (positive reinforcement) (Snyder & Patterson, 1995).

Parents' frequent use of less angry, low-intensity aversive strategies (e.g., nagging, criticism) to influence child behavior is more likely to evoke reciprocal opposition, anger, and P-AGG by the child, especially when these child responses result in frequent parental acquiescence (e.g., cessation of nagging and criticism; leading to escape conditioning). The manner in which aversive family processes facilitate the appearance and growth of COV in early to middle childhood (Patterson, 1982) is less clear. Similar to P-AGG, COV appears to be supported by positive reinforcement (access to socially proscribed but rewarding materials and activities) and negative reinforcement (avoidance of adult tracking and punishment) contingencies (Snyder et al., 2006). The manner in which aversive family environments differentially shape COV and P-AGG may primarily entail the topography of behaviors functionally supported by negative reinforcement. Very angry, harsh, and contingent parental punishment of child oppositional behavior and P-AGG may result in discrimination learning

in which the child's AS behavior is progressively shaped to obtain desired but proscribed materials and activities through surreptitious, covert means (avoidance learning). In response to parental anger and punishment, the child learns to engage in proscribed AS behaviors in ways that minimize or avoid parental tracking and contingencies. As described later, individual differences in children's relative anger and fear reactivity to aversive parental actions and in the executive regulation of anger and fear may also differentially amplify risk for COV and P-AGG.

The capacity for P-AGG appears to be built into human organisms and is apparent very early in development (Tremblay, 2003). In contrast, the later appearance of COV suggests a role for environmental acquisition of COV. The early appearance and growth of COV may result from exposure to social models of deviant behavior and from involvement in conversations that encourage AS behavior, or what has been termed deviancy training. Deviancy training entails obtaining information and encouragement about how, when, and where to engage in COV forms of AS behavior (such as stealing and drug use), how to cover such AS behavior using lying and other surveillance avoidance strategies, and admiration or approval of reports of successful performance of COV activities. Deviancy training has been observed in family and peer environments in middle childhood and is more strongly associated with COV than P-AGG (Snyder, Schrepferman et al., 2005b).

In summary, P-AGG and COV are associated with parents' primary reliance on aversive disciplinary responses to child misbehavior. COV may be the result of a developmental progression in which overt P-AGG, shaped by the experience of parental anger and harsh and contingent punishment, increasingly goes "underground" to COV as children learn to avoid surveillance and consequences adults typically provide for more overt, direct forms of AS behavior. The likelihood of progression to COV depends in part on the nature of parental contingencies for overt defiance and P-AGG. We hypothesized that progression to COV by children showing high rates of early P-AGG will be associated with (1) high rates of parental anger toward the child, (2) parental endorsement and use of harsh discipline tactics, (3) parents' frequent and contingent use of punishment, and also (4) involvement of the child in deviant talk about COV activities during family interaction. In contrast, frequent and persisting P-AGG and reduced risk for progression to early COV will be associated with (1) parents' frequent use of low-intensity, non-contingent aversive but ineffective nagging and "nattering" during interactions with their children, (2) infrequent or low-level parent anger, and (3) parents' frequent acquiescence to the child's overt oppositional behavior and P-AGG. P-AGG and COV also reflect a critical failure by parents to instigate and positively reinforce children's skillful instrumental behavior.

In a broader sense, both P-AGG and COV entail norm-violating responses that engender short-term positive outcomes and avoid or escape from negative environmental contingencies for those responses. These two AS response classes are not incompatible and their relative developmental appearance, co-occurrence, and growth reflect the degree to which they are supported by somewhat different combinations of environmental contingencies and organismic characteristics.

29.5 An Integrative Model of Emotion Regulation, Family Processes, and the Development of Antisocial Behavior

A more complete developmental model for P-AGG and COV can be derived from the integration of emotional, executive control regulatory, and family social learning processes. This model is supported by past research on variables which differentially increment risk for persisting P-AGG and for early onset COV (Loeber and Stouthamer-Loeber, 1998; Patrick, Snyder, Schrepferman, & Snyder,

2005; Stoolmiller & Snyder, 2006). The power of an integrated multivariate model lies in its explanation of how child anger and fear, child executive control, and parent–child discipline interactions make additive and synergistic contributions to risk. An initial multi-process model for P-AGG and COV is now described.

Risk for persisting P-AGG will be facilitated by child anger in response to limit setting (frustrative non-reward) and aversive parent behavior (punishment), especially when child direct oppositional and P-AGG responses often result in parental acquiescence. The facilitating effect of child anger will be even more apparent when counter-balancing fear-based inhibition and anger downregulation by executive control processes are less available. P-AGG entails immediate, direct, angry, counter-coercive child responses to frustrating and aversive environmental events facilitated by (a) the relative absence of sufficient counter-balancing internal inhibition and executive regulation, (b) the occurrence of supportive negative reinforcement (escape) contingencies, and (c) the synergistic interaction of (a) and (b).

Risk for the early appearance and growth of COV will be facilitated when children's early P-AGG in response to limit setting (frustrative non-reward) and aversive parent behavior (punishment) is met with parents' anger and use of contingent, high-intensity aversive reactions. The progression from P-AGG to COV is also facilitated when child anger and immediate counter-coercive reactions are more effectively inhibited by the activation of fear and by executive control processes. The inhibition of immediate, overt counter-coercive behavior and anger enables a discriminated waiting for the absence of environmental monitoring and punishment of proscribed AS behavior (avoidance conditioning) characteristic of COV. Successful planning and concealment of COV acts (promise breaking, lying and surveillance avoidance) are facilitated by adequate executive control. Synergistic interactions among these conditions also contribute to the risk for COV. Angry, harsh parental disciplinary responses with infrequent acquiescence to child overt opposition and P-AGG will increase the risk for COV more powerfully for children with higher, relative to lower, levels of fear and with adequate executive control. Importantly, P-AGG and surreptitious COV are also facilitated by parents' failure to model, cue, and contingently reinforce skillful, normative instrumental responses to manage emotions, attain rewards, and cope with frustrating and aversive environmental events.

These hypotheses are displayed in graphical form in Fig. 29.3. In the figure, the developmental persistence and the growth in P-AGG and COV during middle childhood are represented as vectors. The length of the vectors indicates the degree of persistence or growth in each form of AS during a developmental interval from time 1 to time 2. Vectors which fall toward the horizontal axis represent greater growth in COV, whereas vectors that are more vertical represent greater growth in P-AGG. The upper left panel of the figure represents the hypothesis that a nattering style of discipline facilitates persisting P-AGG (shifts the vectors toward the vertical axis), whereas a harsh style of discipline facilitates growth of COV (shifts the vectors toward the horizontal axis). Good child executive control generally inhibits P-AGG, but its relationship to COV is a curvilinear, inverted U function. At low levels of executive control, there is insufficient inhibition of immediate direct P-AGG in response to aversive environmental events. At an intermediate level of executive control, there is sufficient inhibition and self-monitoring to enable performance of AS responses that avoid environmental surveillance and contingencies – i.e., COV. High levels of executive control may be sufficient to inhibit both COV and P-AGG forms of AS behavior.

The upper right panel of the figure represents the interaction of parental nattering with child trait fear and anger and executive control. Nattering facilitates the persistence of P-AGG more than that of COV and does so more for children evidencing higher trait anger and lower trait fear. Good child executive control reduces the risk (vector length) for persisting P-AGG, given nattering discipline. The lower portion of the figure represents the interaction of harsh discipline with child trait fear and anger. Generally, harsh discipline facilitates growth of COV more than that of P-AGG and does so

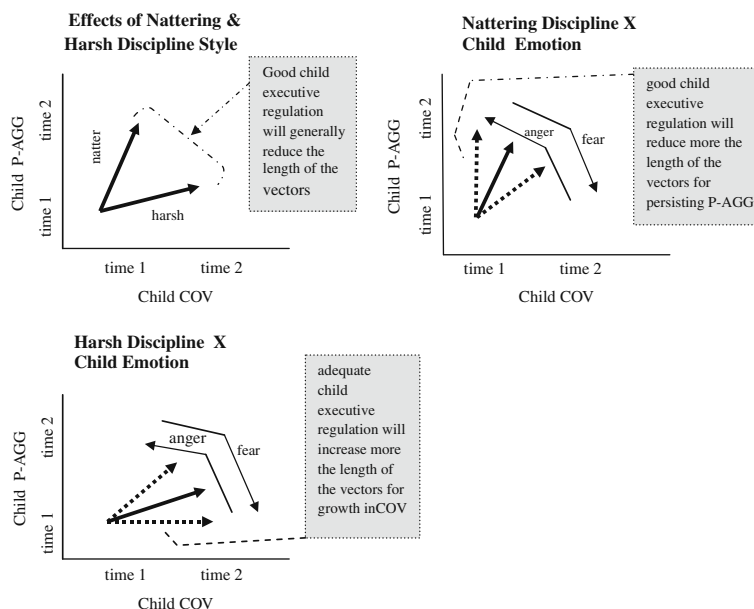


Fig. 29.3 Main and synergistic effects of discipline, child anger and fear, and child executive control regulation on trajectories for physical aggression and covert conduct problems. Note: The length of the vectors in the panels indicates the degree of persistence and growth of child physical aggression (P-AGG) and covert conduct problems (COV) during a developmental interval from time 1 to time 2 during middle childhood. Shifts in vector directions toward the horizontal axis indicate more persistence and growth in COV and toward the vertical axis indicate more persistence and growth in P-AGG. Harsh discipline biases the expression of antisocial behavior toward COV and nattering discipline toward P-AGG. Child anger biases antisocial behavior toward the expression of P-AGG, and fear biases antisocial behavior toward COV. Whereas good executive control generally tends to reduce the risk for antisocial behavior, it has more powerful linear inhibitory effects for P-AGG but has non-linear inverted U effects for COV

more for children evidencing higher trait fear and lower trait anger. Moderately good child executive regulation increases the risk (vector length) for growth in COV, given harsh discipline, but low and high levels of executive control diminish risk for COV.

29.6 An Empirical Test of the Model

The data used to test the proposed model are derived from the School Transitions Project (STP), a longitudinal study of an at-risk community sample of 133 girls and 134 boys from age 5.3 years (fall of kindergarten) to age 7.2 years (spring of first grade). The characteristics of the STP children and families are thoroughly described elsewhere (Patrick et al., 2005; Snyder, Cramer, Afrank, & Patterson, 2005a).

29.6.1 Child P-AGG and COV

These variables were assessed using reliable, validated measures in each of three social ecologies (home: parent ratings, classroom: teacher ratings, and school playground: observations) in the fall

and spring of both kindergarten and first grade. Multi-setting composite scores for P-AGG and COV were calculated at each of four developmental points (fall and spring of both kindergarten and first grade) by rescaling each measure comprising the composite to allow change at a different rate in each setting over time while anchored at a common metric at the initial measurement point. Linear growth models of trajectories of child multi-setting displays of P-AGG and COV during kindergarten and first grade fit the data adequately. P-AGG evidenced significant individual differences in the fall of kindergarten and in growth during kindergarten and first grade. COV also evidenced significant individual differences in the fall of kindergarten and in growth (and average group-level growth) during kindergarten and first grade.

29.6.2 Discipline

Six measures of discipline were derived from 2 h of parent–child interaction observed on each of two occasions during the children’s kindergarten year. This interaction was coded using the family and peer process (FPP) code (Crosby, Stubbs, Forgatch, & Capaldi, 1998) to estimate the rate per minute at which parents directed low-intensity aversive behavior (nagging and *nattering*) toward their child, the proportion of occasions on which parents *acquiesced* to their children’s counter-coercive behavior during discipline episodes, and the occurrence of family *deviant talk* (see Snyder, Schrepferman et al., 2005b). FPP coders also made repeated ratings of parent *inconsistent discipline* tactics (see Snyder et al., 2005a). A second set of assessors coded parent–child interaction using the specific affect (SPAFF) code (Gottman, McCoy, Coan, & Collier, 1996; see Snyder, Stoolmiller, Wilson, & Yamamoto, 2003 for details). This SPAFF coding was used to estimate rates at which parents displayed *anger* toward their child. A sixth measure assessed parents’ self-reported use of *harsh discipline* tactics (e.g., spanking, threats) in response to common problem child behavior (Snyder et al., 2005a).

Bivariate correlations among the measures of discipline are shown in Table 29.1. There was sufficient convergence among the measures to create a multi-indicator discipline construct. However, most of the correlations were also modest in size, suggesting that discipline is a complex, multi-dimensional construct. Therefore, the relation of each of the discipline indicators taken one at a time as well as the relation of a multi-indicator discipline construct to growth in P-AGG and COV were examined using SEM.

Table 29.1 Correlations among discipline measures

	Natter	Acquiesce	Inconsistent	RPM anger	Deviant talk
Natter					
Acquiesce	0.25**				
Inconsistent	0.29**	0.22**			
RPM anger	0.34**	0.19*	0.27**		
Deviant talk	0.18*	0.02	0.36**	0.28**	
Harsh tactics	0.17*	0.19*	0.23**	0.27**	0.24**

* $p < 0.05$; ** $p < 0.01$

29.6.3 Discipline and P-AGG and COV

The results of SEM models testing the relation of each measure of discipline to child P-AGG and COV in the fall kindergarten and to growth in P-AGG and COV during kindergarten and first grade

Table 29.2 The relationship of discipline to child physical aggression and covert conduct problems

Parenting factor	Physical aggression		Covert conduct problems	
	Fall K	Growth	Fall K	Growth
RPM nattering	0.21**	0.17	0.06	0.11
Parent acquiesce	0.27***	0.18	0.01	−0.07
Inconsistent discipline	0.22**	0.10	0.25**	0.38**
RPM parent anger	0.10	0.12	0.21**	0.34**
Family deviant talk	0.07	0.19	0.22**	0.36**
Endorse harsh tactics	0.03	0.12	0.19**	0.26**

* $p < 0.05$; ** $p < 0.01$; *** $p < 0.001$

Note: Parameters shown in bold are significantly different for physical aggression and covert conduct problems.

are shown in Table 29.2. Parents' rate of low-level aversive nattering, acquiescence to aversive child behavior, and inconsistent discipline were significantly associated with higher rates of multi-setting child P-AGG in the fall of the kindergarten year. Neither nattering nor acquiescence was related to COV in the fall of kindergarten. In contrast, observed rates of parent anger toward the child, children's observed exposure to deviant talk during parent–child interaction, parents' endorsement of harsh discipline tactics, and inconsistent discipline were associated with higher multi-setting displays of COV in the fall of kindergarten and with growth in COV during kindergarten and first grade, but not with growth in physical aggression. Many of the relationships of the various measures of discipline to P-AGG and COV were reliably different (shown in bold in Table 29.2). This pattern of findings replicates previous research indicating the role of ineffective discipline in the development of AS during childhood (Patterson, Reid, & Dishion, 1992). However, it also suggests a more differentiated set of empirical relationships consistent with the hypothesis that highly coercive discipline (indicated by parental anger and harsh tactics) is more powerfully related to the early appearance and growth of COV, whereas parental nattering and acquiescence to child coercive behavior are more powerfully related to P-AGG.

29.7 Child Emotional Reactivity and Frontal Executive Regulation

Four child characteristics were measured in kindergarten: inhibition and verbal skills (executive control) and trait fear and anger displays (emotional reactivity). *Inhibition* was defined by a composite derived from the Trails Test B, WISC-R digit span, observed on-task academic engaged time in the classroom, and assessors' ratings of children's attention during individual assessment tasks (see Snyder, Prichard, Schrepferman, Patrick, & Stoolmiller, 2004). *Verbal ability* was measured by the Peabody Picture Vocabulary Test – Revised (PPVT; Dunn & Dunn, 1981). Trait *anger and fear reactivity* were each defined by averaging children's observed rates of anger and of fear during 1 h of interaction with parents on each of two occasions and during 15 min of interaction with same gender classmates on each of three occasions. Anger and fear during these interactions were coded using SPAFF (Gottman, McCoy, Coan, & Collier, 1996).

The bivariate correlations between child trait anger and fear reactivity, inhibition and verbal ability (see Table 29.3) were small and often insignificant. The one exception was the large positive relationship between inhibition and verbal skills, both of which represent executive control. Trait anger and fear reactivity were positively correlated at a modest level, perhaps reflecting a general negative emotionality. Child verbal skills and inhibition were not strongly related to anger and fear

Table 29.3 Correlations among child emotion traits and self-regulatory capacities

	Inhibition	Verbal skills	Trait anger
Inhibition			
Verbal skills	0.52**		
Trait anger	0.05	−0.06	
Trait fear	−0.14*	−0.16*	0.21**

* $p < 0.05$; ** $p < 0.01$.

reactivity, indicating that separate exploration of their relationships to child P-AGG and COV will not be impeded by highly redundant results.

The relationships of these child characteristics to P-AGG and COV in fall kindergarten and to growth in P-AGG and COV during kindergarten and first grade were tested in SEM and are shown as standardized coefficients in Table 29.4. Children high in anger reactivity were more likely to show growth in both P-AGG and COV. Children high in trait fear reactivity were less likely to evidence persistence or growth in P-AGG but were more likely to show growth in COV, and this difference in the association of fear reactivity with growth in P-AGG and COV was significant. Children with executive control inhibitory capacities and verbal skills were less likely to evidence P-AGG and COV in fall kindergarten. These findings are consistent with previous research indicating that child anger increases the risk for AS behavior and that executive control as indexed inhibition and verbal competence decreases that risk. More important for the proposed model, fear reactivity was associated with less growth in P-AGG and more growth in COV.

Table 29.4 The relationship of child characteristics to individual differences in physical aggression and covert conduct problems

Child characteristic	Physical aggression		Covert conduct problems	
	Fall K	Growth	Fall K	Growth
Trait anger reactivity	0.01	0.20*	−0.05	0.33**
Trait fear reactivity	0.04	−0.35***	−0.03	0.21**
Inhibition (executive control)	−0.28**	−0.05	−0.27**	−0.14
Verbal skills	−0.21*	−0.09	−0.16*	−0.25*

* $p < 0.05$; ** $p < 0.01$; *** $p < 0.001$

Note: Parameters shown in bold are significantly different for physical aggression and covert conduct problems.

29.8 Tests of the Integrated Model

The final models test the additive and synergistic contributions of child characteristics (trait anger and fear reactivity, and executive control as indexed by inhibition and verbal skills, each taken one at a time) and ineffective parent discipline (represented as a construct using all six indicators shown in Table 29.2) to child P-AGG and COV. The use of this multi-indicator construct of *ineffective discipline* has both advantages and disadvantages. One disadvantage is that various indicators do not seem to be equivalent predictors of P-AGG and COV (see Table 29.2). However, the indicators are reliably correlated and a SEM confirmatory factor analysis showed that they all loaded significantly

and at comparable levels on an ineffective discipline construct. The use of a multi-indicator construct is advantageous because it provides a parsimonious approach to testing discipline in the integrated model. The strong measurement qualities of the discipline construct also provide a stringent empirical test of the relation of child emotional reactivity (trait anger and fear) and executive regulatory capacity (inhibition and verbal skill) to P-AGG and COV because the predictive value of each child characteristic competes statistically with the robust ineffective discipline construct.

A separate model was fit to the data to assess the additive and synergistic contribution of ineffective discipline and each of the child characteristics (taken one at a time) to P-AGG and COV. The results of SEM tests of these models are shown in Table 29.5. Each major horizontal section in the table represents a separate SEM model incorporating the simultaneous contribution of ineffective discipline, one child characteristic, and the interaction of ineffective discipline and that child characteristic to P-AGG (left two data columns) and COV (right two data columns). The table provides standardized path coefficients and the amount of variance accounted for in fall kindergarten levels and growth of P-AGG and COV during kindergarten and first grade.

Table 29.5 Relationships of ineffective discipline, child anger and fear, executive inhibition, and verbal skills to multi-setting physical aggression and covert conduct problems

Predictors	Physical aggression		Covert conduct problems	
	Fall K	Growth	Fall K	Growth
Ineffective discipline	0.21*	0.17	0.32**	0.47**
Trait fear reactivity	0.04	-0.21*	0.03	0.39**
Discipline X fear	0.01	0.02	0.05	0.17
R ²	0.05	0.12	0.11	0.41
Ineffective discipline	0.20*	0.18	0.32**	0.44**
Trait anger reactivity	-0.02	0.26*	0.02	0.25*
Discipline X anger	0.01	-0.04	0.03	-0.06
R ²	0.04	0.10	0.10	0.26
Ineffective discipline	0.18*	0.11	0.28**	0.44**
Inhibition (executive)	-0.35**	-0.05	-0.25**	-0.21*
Discipline X inhibition	0.10	0.33**	0.06	0.04
R ²	0.10	0.16	0.14	0.23
Ineffective discipline	0.19*	0.10	0.31*	0.43**
Verbal skills	-0.29**	-0.09	-0.07	-0.18
Discipline X verbal skills	-0.01	0.21*	0.07	-0.23*
R ²	0.12	0.06	0.11	0.27

* $p < 0.05$; ** $p < 0.01$

Note: Parameters shown in bold are significantly different for physical aggression and covert conduct problems. See text for interpretation of interaction effects.

29.8.1 Main Effects of Discipline

Ineffective discipline was associated with higher fall kindergarten levels of both P-AGG and COV in models including each of the child characteristics. Ineffective discipline reliably predicted the growth of COV but not P-AGG during kindergarten and first grade. The relation of ineffective discipline to growth in COV was significantly greater than that to growth in P-AGG. Ineffective discipline appears

to increase the risk of both forms of antisocial behavior but may play a particularly critical role in the transition to COV during middle childhood.

29.8.2 Main Effects of Child Anger, Fear, and Executive Control

Children's emotional reactivity, capacity for executive inhibition, and verbal skills were related to trajectories for P-AGG and COV in both similar and unique ways. Fear reactivity was unrelated to kindergarten P-AGG and COV. Fear reactivity was negatively related to growth in P-AGG ($b = -0.21$) but positively related to growth in COV ($b = 0.39$). Children's propensity to experience and display fear in response to aversive environmental events such as harsh parental discipline may facilitate a developmental progression or a transformation in AS behavior. This transition may entail a move from primary reliance on direct P-AGG to gain rewards or terminate conflict to the increasing use of COV tactics in response to limit setting and punishment in order to simultaneously gain rewards and avoid parental tracking and negative consequences for norm-violating behavior. In the relative absence of fear, direct and confrontational overt tactics such as P-AGG may persist.

Children's trait anger reactivity was related to the growth of both P-AGG and COV. Anger may facilitate mobilization of both overt and covert AS responses to attain rewards in the face of environmental constraints and potential punishment. Replicating previous research, child executive control, and verbal skills were generally associated with reduced risk of P-AGG and COV in early kindergarten and with reduced growth of COV during kindergarten and first grade.

29.8.3 Synergistic Effects of Discipline and Child Self-regulation

The third row of data in the sections for each model in Table 29.5 indicates the degree to which each child characteristic (taken one at a time) and ineffective discipline are synergistically associated with trajectories of P-AGG and COV. The interactions of ineffective discipline with anger reactivity and with fear reactivity were not significantly associated with trajectories of either P-AGG or COV. It may be that emotional reactivity mediates rather than moderates the relationship of ineffective discipline to AS development (Snyder et al., 2003). The interaction of ineffective discipline and executive inhibition was significantly associated with growth of P-AGG. Ineffective discipline appears to facilitate the growth of P-AGG most powerfully for those children with poor executive control, in addition to the main effects of discipline and inhibition. The interaction of discipline and executive inhibition was not associated with growth in COV.

The interaction of ineffective discipline and verbal skill was significantly associated with growth in both P-AGG and COV, but opposite ways. Ineffective discipline was more powerfully associated with growth in P-AGG for children with lower verbal skills but was more powerfully associated with growth in COV for children with higher verbal skills, in addition to the main effects of discipline and verbal skills.

29.8.4 Summary of Findings

The child characteristics and family conditions associated with increased risk for child P-AGG and COV when children are beginning kindergarten appear to be quite similar and are consistent with risk factors associated with early onset antisocial behavior in previous research: ineffective discipline, high child anger reactivity, and poorly developed child executive control. The child characteristics

and family conditions associated with growth in P-AGG and COV over the next 18 months, however, were quite different. Growth in COV relative to P-AGG appears to be more strongly facilitated by poor and perhaps more highly coercive and angry parental discipline, especially for children with better verbal skills and adequate executive control. This supports the hypothesis that children who have adequate verbal skills and executive control “go underground” toward more covert expressions of AS behavior when faced with ineffective, aversive parental discipline. Adequate executive control, higher fear reactivity, and good verbal skills may facilitate behavioral inhibition, discriminated waiting, and planning, all of which facilitate the adept performance of delayed and surreptitious (covert) AS behavior to attain desired outcomes while avoiding surveillance and corrective social contingencies.

On the other hand, growth in P-AGG relative to COV appears to be facilitated by lower child fear reactivity and by poor child executive inhibition and verbal skills along with an ineffective parent discipline style characterized by nattering and acquiescence to children’s direct, overt counter-coercion. Children with persisting P-AGG have less capacity to inhibit immediate and direct confrontational responses to aversive and frustrating environmental events, and experience fewer environmental contingencies that encourage behaving in more surreptitious ways.

29.9 Research on Emotions: Implications, Extensions, and New Opportunities

The application of the multi-process model to the longitudinal STP data provided an increased understanding of the development and etiology of P-AGG and COV. The persistence of early P-AGG was the result of one combination of social, emotional, and regulatory processes: a nattering discipline style, parent acquiescence to child aggressive behavior, high child anger reactivity, low child fear reactivity, and low child verbal and executive control capacities. However, P-AGG was increasingly overlaid with and transformed into COV as the result of another combination of social, emotional, and regulatory processes: frequent parent anger and reliance on harsh discipline tactics, especially when applied to children with higher fear reactivity and with adequate verbal skills and executive control. Consistent with the integrated model, emotional and social processes had both additive and synergistic effects on development. Progressions and transformations in the expression of AS behavior appear to reflect ongoing adaptation to unskilled and aversive parenting conditional on the affective and executive self-regulatory characteristics of the child. The more general utility of the proposed model awaits its replication and extension to other forms of psychopathology.

29.9.1 Implications

The success of the model in accounting for variations in the development of AS behavior may have more general implications for theory and research on anger, fear, and other emotions. An exclusive focus on specific emotions (such as anger) in isolation from other emotions and from other neural-regulatory, behavioral, and environmental processes may lead to a limited understanding of how emotions contribute to adaptation and maladaptation. The frequency, the intensity, and the duration of any one emotion are likely to be shaped by the functional properties (environmental consequences) of the behaviors it engenders *relative to* the functional properties of behaviors engendered by other emotions. For example, anger-supported direct counter-attack (P-AGG) appeared to be functional for children whose parents engaged in discipline characterized by nattering and acquiescence to direct child coercion. Anger-supported, direct counter-attack appeared to be less functional for children whose parents engaged in discipline characterized by frequent anger and contingent forms of harsh

punishment. In the latter discipline environment, inhibition of immediate and direct counter-attack and activation of fear in response to parental anger and punishment was quite functional and shaped discriminated waiting to engage in norm-violating behavior when adult monitoring is diminished and adult contingencies were less likely – that is, COV forms of AS behavior.

29.9.2 Extensions

Research on emotions (including the data presented in this chapter) has often relied on “trait-like” theoretical and methodological approaches to examine how arousal and regulation of anger and other emotions contribute to risk for psychosocial problems. These trait approaches often couch emotional reactivity and emotion regulation in terms of temperament or personality constructs and measure those constructs using global reports or ratings that average emotional responses over substantial time periods and across a broad range of situations. However, trait approaches provide little information about how emotional responses and regulation evoke environmental reactions or are progressively shaped by environmental experiences in ways that imbue risk for maladaptive behavior. In contrast to trait approaches, the conserved and adaptive functions of emotions are most apparent in their situation-specific and time-dynamic responsiveness to salient cues for environmental rewards and threats of punishment. From this perspective, emotional arousal and regulation, and their reciprocal relationships to overt behavior and environmental events need to be construed in a more dynamic perspective: emotions are rapidly engendered in response to shifts in salient organismic and environmental opportunities and challenges, and dissipate relatively quickly as those opportunities and challenges are accommodated behaviorally and physiologically (Chapter 22). From this perspective, the activation and regulation of emotions should be sensitive to their immediate functional properties.

As an example of this alternative functionalist approach, repeated-events, competing-risk event history analyses were applied to real-time observed parent–child interaction data from the STP study (Dagne & Snyder, 2007; Snyder et al., 2003; Stoolmiller & Snyder, 2006). These analyses indicated that the occurrence of aversive parent behavior substantially reduced the latencies (time intervals) for a child to move from a neutral emotional state to anger or to fear. Focusing on anger specifically, children were quicker to re-express anger as parents responded in hostile ways to children’s expressions of anger, and each hostile parental response increased the hazard of children’s re-expression of anger (c.f., anger priming effect, Chapter 22).

The temporal dynamics and functions of child anger during parent–child interaction were associated with children’s P-AGG and COV in different ways. Child P-AGG was associated with longer duration child displays of anger in response to aversive parent behavior and with an increasing likelihood that parents would then shift to a non-aversive behavior (i.e., to acquiesce to child anger). P-AGG was also associated with increasingly longer duration displays of child anger as those displays accumulated during ongoing parent–child interaction. In contrast, child COV was associated with children’s more rapid desistance from anger when their parents were hostile or aversive and with decreasing durations of child anger displays as those displays were repeated during ongoing parent–child interaction. The results of these micro-level analyses complement those derived from the trait-like analyses described in this chapter but apply a more powerful lens to the proximal bilateral functions that anger serves in ongoing parent–child interaction.

29.9.3 New Opportunities

More recently, a number of methodological advances have provided the means to measure the functional properties, brain emotion, and regulatory networks in reliable and objective ways (e.g., fMRI

and dense array EEG indices of localized brain activity and closely associated neuropsychological marker tasks). These advances provide the opportunity to incorporate carefully measured and precisely specified organismic variables into empirically testable dynamic, multi-process systems models in ways that wed the strengths of functionalist, social learning approaches with the strengths and richness of affective neuroscience, neurophysiology, and molecular genetics. The challenge is to articulate system models that incorporate and integrate the reciprocal interplay of environmental events and overt behaviors (and their contingent relationships), and executive, verbal, and motivation–emotion systems by which organisms activate and guide behavior in the context of salient environmental events. An even greater challenge will be to examine the interplay of these multi-process systems from a developmental perspective. This interplay is likely to be transactional, reflecting how behavior–environment contingencies shape intra-organismic emotional–motivational and regulatory networks, and how variation in the functional properties of these networks (as a result of genetic variation, biological maturation, and cumulative shaping by experience) engender and shape physiological and behavioral responses to salient environmental events.

References

- Bongers, I. L., Koot, H. M., van der Ende, J., & Verhulst, F. C. (2003). The normative development of child and adolescent problem behavior. *Journal of Abnormal Psychology, 112*, 179–192.
- Broidy, L. M., Nagin, D. S., Tremblay, R. E., Bates, J. E., Brame, B., Dodge, K. A., et al. (2003). Developmental trajectories of childhood disruptive behaviors and adolescent delinquency: A six-site, cross-national study. *Developmental Psychology, 39*, 222–245.
- Bugental, D. B., & Grusec, J. E. (2006). Socialization processes. In W. Damon, R. M. Lerner, & N. Eisenberg (Eds.), *Handbook of child psychology: Vol. 3: Social, emotional and personality development* (6th ed.). Hoboken, NJ: John Wiley & Sons.
- Crosby, L., Stubbs, J., Forgatch, M. S., & Capaldi, D. (1998). *Family and peer process code training manual*. Eugene, OR: Oregon Social Learning Center.
- Dagne, G. A., & Snyder, J. (2007). *Bayesian analysis of repeated behaviors using event-dependent frailty models*. Unpublished manuscript, University of South Florida.
- Derryberry, D., & Tucker, D. M. (2006). Motivation, self-regulation, and self organization. In D. Cicchetti & D. J. Cohen (Eds.), *Developmental psychopathology. Vol. 2: Developmental neuroscience* (2nd ed., pp. 502–533). Hoboken, NJ: John Wiley & Sons.
- Dishion, T. J., & Patterson, G. R. (2006). The development and ecology of antisocial behavior in children and adolescents. In D. Cicchetti & D. J. Cohen (Eds.), *Developmental psychopathology. Vol. 3: Risk, disorder, and adaptation* (2nd ed., pp. 503–541). Hoboken, NJ: John Wiley & Sons.
- Dunn, L. M., & Dunn, L. M. (1981). *Peabody picture vocabulary test manual*. Circle Pines, MN: American Guidance Service.
- Ekman, P., & Davidson, R. J. (1994). *The nature of emotion: Fundamental questions*. New York: Oxford University Press.
- Frick, P. J., Lahey, B. B., Loeber, R., Tannenbaum, L., Van Horn, Y., Christ, M. A. G., et al. (1993). Oppositional defiant disorder and conduct disorder: A meta-analytic review of factor analyses and cross-validation in a clinic sample. *Clinical Psychology Review, 13*, 319–340.
- Gottman, J. M., McCoy, K., Coan, J., & Collier, H. (1996). *The specific affect coding system (SPAFF) for observing emotion communication in marital and family interaction*. Mahwah, NJ: Erlbaum.
- Gray, J. A. (1994). Neural systems, emotion and personality. In J. Madden, S. Mattheysee, & J. Barchas (Eds.), *Adaptation, learning and affect* (pp. 95–121). New York: Raven Press.
- Hinshaw, S. P., Simmel, C., & Heller, T. L. (1995). Multimethod assessment of covert antisocial behavior in children: Laboratory observations, adult ratings, and child self report. *Psychological Assessment, 7*, 209–219.
- Izard, C. E. (1991). *The psychology of emotions*. New York: Plenum.
- Izard, C. E. (2002). Translating emotion theory and research into preventive interventions. *Psychological Bulletin, 128*, 796–824.

- Lahey, B. B., & Waldman, I. D. (2003). A developmental propensity model of the origins of conduct problems during childhood and adolescence. In B. B. Lahey, T. E. Moffitt, & A. Caspi (Eds.), *Causes of conduct disorder and juvenile delinquency* (pp. 76–117). New York: Guilford Press.
- Lewis, M., & Haviland-Jones, J. M. (2000). *Handbook of emotions* (2nd edition). New York: Guilford Press.
- Loeber, R., Keenan, K., & Zang, Q. (1997). Boy's experimentation and persistence in developmental pathways toward serious delinquency. *Journal of Child and Family Studies*, 6, 321–357.
- Loeber, R., & Stouthamer-Loeber, M. (1998). Development of juvenile aggression and violence: Some common misconceptions and controversies. *American Psychologist*, 53, 242–259.
- Luciana, M. (2006). Cognitive neuroscience and the prefrontal cortex: Normative development and vulnerability to psychopathology. In D. Cicchetti & D. J. Cohen (Eds.), *Developmental psychopathology. Vol. 2: Developmental neuroscience* (2nd ed., pp. 292–331). Hoboken, NJ: John Wiley & Sons.
- Moffitt, T. E., Lynam, D., & Silva, P. (1994). Neuropsychological tests predict persistent male delinquency. *Criminology*, 32, 101–124.
- Panksepp, J. (1998). *Affective neuroscience*. New York: Oxford University Press.
- Patrick, M. R., Snyder, J., Schrepferman, L. M., & Snyder, J. M. (2005). The joint effects of early parental warmth and communication and child conduct problems on later monitoring and child conduct problems. *Child Development*, 76, 999–1014.
- Patterson, G. R. (1982). *Coercive family process*. Eugene, OR: Castalia Publishing.
- Patterson, G. R. (1993). Orderly change in a stable world: The antisocial trait as chimera. *Journal of Consulting and Clinical Psychology*, 61, 911–919.
- Patterson, G. R., Reid, J. B., & Dishion, T. J. (1992). *Antisocial boys*. Eugene, OR: Castalia Publishing.
- Posner, M. I., & Rothbart, M. K. (2007). *Educating the human brain*. Washington, DC: American Psychological Association.
- Rothbart, M. K., Posner, M. I., & Kieras, J. (2006). Temperament, attention, and the development of self-regulation. In K. McCartney, & D. Phillips (Eds.), *The Blackwell handbook of early childhood development* (pp. 328–357). London: Blackwell Press.
- Rubin, K. A., Bukowski, W. M., & Parker, J. G. (2006). Peer interactions, relationships and groups. In W. Damon, R. M. Lerner, & N. Eisenberg (Eds.), *Handbook of child psychology. Vol. 3. Social, emotional and personality development* (6th ed.). Hoboken, NJ: John Wiley & Sons.
- Snyder, J. (2002). Reinforcement and coercion mechanisms in the development of antisocial behavior: Peer processes. In J. R. Reid, G. R. Patterson, & J. Snyder (Eds.), *Antisocial behavior: Prevention, intervention and basic research* (pp. 101–122). Washington, DC: American Psychological Association Press.
- Snyder, J., Cramer, D. A., Afrank, J., & Patterson, G. R. (2005a). The contribution of ineffective discipline and parent hostile attributions about child misbehavior to the development of conduct problems at home and school. *Developmental Psychology*, 41, 1–12.
- Snyder, J., Edwards, P., McGraw, K., Kilgore, K., & Holton, A. (1994). Escalation and reinforcement in mother-child conflict: Social processes associated with the development of physical aggression. *Development and Psychopathology*, 6, 305–321.
- Snyder, J., McEachern, A., Schrepferman, L., Zettle, R., Johnson, K., Swink, N., et al. (2006). Rule governance, correspondence training and discrimination learning: A developmental analysis of covert conduct problems. *Speech and Language Pathology and Applied Behavior Analysis*, 1, 43–54.
- Snyder, J., & Patterson, G. R. (1995). Individual differences in social aggression: A test of a reinforcement model of socialization in the natural environment. *Behavior Therapy*, 26, 371–391.
- Snyder, J., Prichard, J., Schrepferman, L., Patrick, R., & Stoolmiller, M. (2004). Child hyperactivity-impulsivity-inattention and verbal-cognitive skills, early peer experiences, and the development of antisocial behavior. *Journal of Abnormal Child Psychology*, 32, 579–594.
- Snyder, J., Schrepferman, L., Oeser, J., Patterson, G. R., Stoolmiller, M., Johnson, K., et al. (2005b). Peer deviancy training and affiliation with deviant peers in young children: Occurrence and contribution to early onset conduct problems. *Development and Psychopathology*, 17, 397–413.
- Snyder, J., & Stoolmiller, M. (2002). Reinforcement and coercion mechanisms in the development of antisocial behavior: Family processes. In J. R. Reid, G. R. Patterson, & J. Snyder (Eds.), *Antisocial behavior: Prevention, intervention and basic research*. Washington, DC: American Psychological Association Press.
- Snyder, J., Stoolmiller, M., Wilson, M., & Yamamoto, M. (2003). Child anger regulation, parental responses to children's anger displays, and early child antisocial behavior. *Social Development*, 12, 335–360.
- Stoolmiller, M. (1992). *Differences between families in extended chains of coercive behavior*. Unpublished manuscript, Oregon Social Learning Center, Eugene, OR.

- Stoolmiller, M., & Snyder, J. (2006). Modeling heterogeneity in social interaction processes using multi-level survival analysis. *Psychological Methods*, 26, 164–177.
- Tremblay, R. E. (2003). Why socialization fails: The case of chronic physical aggression. In B. B. Lahey, T. E. Moffitt, & A. Caspi (Eds.), *Causes of conduct disorder and juvenile delinquency* (pp. 182–224). New York: Guilford Press.
- Verhulst, F. C., Koot, H. M., & Berden, G. F. (1990). Four year follow-up of an epidemiological sample. *Journal of the American Academy of Child and Adolescent Psychiatry*, 29, 440–448.
- Willoughby, M., Kupersmidt, J., & Bryant, D. (2001). Overt and covert dimensions of antisocial behavior in early childhood. *Journal of Abnormal Child Psychology*, 29, 177–187.

Chapter 30

Anger in Intimate Relationships

Donald G. Dutton

If a woman loses her beauty, or shows that she doesn't have the strength and dependability that we once thought she did, or loses her intellectual sharpness, or falls short of our own peculiar needs in any of a thousand ways, then all the investment we have made in her is undermined. The shadow of imperfection falls over our lives and with it- death and the defeat of cosmic heroism. ... this is the reason for so much bitterness, shortness of temper and recrimination in our daily family lives. ... We may have no other God and we may prefer to deflate ourselves in order to keep the relationship, even though we glimpse the impossibility of it and of the slavishness to which it reduces us.

– Ernest Becker: The Denial of Death, 1973, p. 167.

Abstract I review studies of anger in intimate relationships: both the heightened incidence rates found and the motivational origin. While high levels of anger are reported in intimate relationships, this anger seems to be part of a more pervasive personality pattern that has heightened reactivity to real or symbolic abandonment – variously called “negative emotionality” or borderline personality. I characterize intimacy anger as a vestige of attachment insecurity and as an over-reactive, occasionally dysfunctional activation of the attachment behavioral system. This appears to have origins in temperament and parental rejection as well as parental abuse. This attachment origin appears to crystallize into a chronic personality disorder in some people, who then become at risk for intimate partner violence (IPV).

30.1 Anger in Intimate Relationships

Intimate relationships, perhaps because they have so much meaning regarding one's sense of selfhood or self-definition (Becker, 1973), reliably generate strong emotional reactions in people. One of these reactions is anger. Even the anticipation of exposure to an intimate conflict is sufficient to increase anger levels in most people (Strachan & Dutton, 1992; Thomas & Dutton 2004). Conflict in intimate relationships (Coleman & Straus 1992) generates high levels of anger and physical attacks, especially when a non-consensual power imbalance exists between the two intimate dyad members. It is possible that anger found in intimate relationships, especially those that generate physical aggression, is a product of the individuals comprising the dyad, is generated by the dyadic conflict, or both.

D.G. Dutton (✉)

Department of Psychology, University of British Columbia, Vancouver, BC, Canada

e-mail: dondutton@shaw.ca

30.2 Trait Anger in Intimately Violent Males

Using the Buss and Durkee (1957), Maiuro, Cahn, Vitaliano, Wagner, and Zegree (1988) found that a sample of domestically violent men had significantly higher levels of both anger and hostility than did controls. The authors concluded that their findings supported the “idea that anger dyscontrol is a key issue in the profile of domestically violent men” (p. 17) and noted that depression, as well as anger, was elevated in this group. They did not, however, speculate on the specific nature of the anger–depression relationship, i.e., whether depression generates anger as an escape from depression or whether both are symptoms of a deeper personality-affective style.

Margolin, John, and Gleberman (1989) found that physically aggressive husbands reported significantly higher levels of anger than did control group husbands. Dutton and Browning (1988) showed videotaped husband–wife conflicts to males convicted of wife assault and control males. The assaultive males reported significantly higher levels of anger than did controls to all male–female conflicts but especially to an “abandonment” scenario, where a woman tells her husband she is visiting another city with women friends for a holiday. The assaultive males perceived more abandonment in this scenario and reported more anxiety as well as anger. This finding raised the possibility of a cognitive-affective template of fear and anger generated by intimacy and relationship loss in abusive men. In all analogue studies of emotional reactions to intimate conflicts, fear and anger were positively and significantly correlated (Strachan & Dutton, 1992; Dutton, Webb, & Ryan, 1994; Jack, Dutton, Webb, & Ryan, 1995; Thomas & Dutton, 2004). Moffitt, Caspi, Rutter and Silva (2001) described an emotional template, which they called “negative emotionality” or “NEM” in abusive women. NEM was composed of ineffective stress reactions, strong emotions such as anger and anxiety, mistrust of people, and a value on vengeance and was related to the use of violence against male partners independent of whether the male used intimate violence. In a review of studies assessing anger and hostility in court-mandated samples of partner-violent men, Holtzworth-Munroe, Bates, Smutzler, and Sandin (1997) reported that all but one study found anger elevations in assaultive men and the exception study had an unacceptably small sample.

30.3 Anger as an Attachment Response

Sonkin and Dutton’s application of attachment theory to intimate violence (Dutton and Sonkin 2003) viewed insecure attachment patterns as essentially maladaptive methods of regulating affect, particularly anger and other emotions stemming from loss. Dutton, Saunders, Starzomsik, and Bartholomew (1994) found elevated anger in assaultive males to be related to certain attachment disorders, especially an attachment style called “fearful” attachment and which they re-labeled “fearful-angry” attachment. They cited Bowlby’s (1977) work on attachment that viewed anger as having a developmentally primary purpose of signaling and seeking re-unification with the attachment object. Hence, dysfunctional anger in adult intimate relationships was viewed by attachment theory as anger expression that served to further distance the attachment object (such as anger-motivated withdrawal or abuse). Dutton et al. argued that the profile of anxious attachment combined with male sex role conditioning to produce the angry and controlling aspect of the fearful-angry attachment style. However, other researchers have found attachment–anger in both genders to be related to abusiveness (Follingstad, Bradley, Helff, & Laughlin, 2002; Mauricio, Tein, & Lopez, 2007). Dutton et al. (1994) explored the developmental origins of elevated anger in assaultive males, viewing it as being produced by a combination of paternal rejection, exposure to interparental physical abuse, and a failure of protective attachment. This combination of experiences in the family of origin produced

chronic trauma symptoms and PTSD – profile similar to that found in Vietnam veterans and that included chronic anger (Dutton 1995a, b; Dutton and Holtzworth-Munroe 1997; Dutton 1999).

In the Follingstad et al. (2002) study, insecure attachment and angry temperament were examined in a large sample of college dating relationships. Their resulting model found that anxious attachment related to angry temperament which in turn generated control. Control in turn generated violence. As the authors put it “the primary path leading to the use of force in dating relationships was initially due to the presence of anxious attachment influencing the development of an angry temperament which then leads to behaviors to control one’s partner” (p. 42).

The controlling behavior was the significant mediator between the angry temperament and the greater frequency and severity of dating violence. Hence, the angry temperament manifested itself in control of the partner which sometimes produced physical violence as a control technique. Attachment was predictive of control and physical abuse by both genders.

Mauricio et al. (2007) also tested the notion that attachment was related to abusiveness in a sample of men in a court-mandated treatment program for spouse assault. The men completed a battery of questionnaires assessing attachment style (i.e., the Experiences in Close Relationships Questionnaire), antisocial and borderline personality disorders (the Personality Disorder Questionnaire), and abusiveness [the Conflict Tactics Scale or CTS (Straus, 1992) and Psychological Maltreatment of Women Scale or PMWI (Tolman, 1989)]. Using path analysis, the authors found that anxious attachment and borderline personality disorder scores were highly correlated. Anxious attachment was also correlated with antisocial personality scores. In other words, it appeared that the personality disorder mediated the effects of attachment insecurity on abuse. The insecure attachment had crystallized into a personality disorder and manifested itself on abusiveness through the personality disorder. Both personality disorders observed in this study had anger as a central feature. Since personality disorder is a major risk factor for abusiveness (Dutton 1994a, b; Moffitt et al., 2001; Ehrensaft, Moffitt, & Caspi, 2004; Ehrensaft, Cohen, & Johnson, 2006), studies found that a personality disorder–abuse relationship may have been capturing a masked attachment disorder. Hence, several studies on psychological profiles of perpetrators of intimate abuse support Bowlby’s notion of the centrality of attachment as a basic human motive whose frustration generates extreme anger (Bowlby, 1969, 1973; Bowlby, 1980).

30.4 Anger as a Symptom of Personality Disorder

Borderline personality organization (BPO) (Gunderson, 1984) is a clinical category characterized by intense anger and impulsivity, unstable interpersonal relationships, and a sense of self. BPO is a less severe form of the more rare borderline personality disorder (Kernberg, 1977). The difference is that BPO is characterized as a continuum running from low to high borderline traits (see also Westen & Shedler, 1999), whereas BPD is a distinct category.

As Gunderson (1984) describes the borderline personality (BP), the essential characteristics (in order of importance) are as follows: a proclivity for intense, unstable interpersonal relationships characterized by intermittent undermining of the significant other, manipulation, and masked dependency; an unstable sense of self with intolerance of being alone and abandonment anxiety; and intense anger, demandingness, and impulsivity, usually tied to substance abuse or promiscuity (c.f., Chapter 27 by R.W. Novaco, this book.)

Gunderson described a three-phase defense sequence of BPO that produces sudden shifts in “phenomenology,” affect, and behavior. This defense sequence could theoretically produce the kinds of behavior depicted by Walker’s (1979) “abuse cycle” description of some wife assaulters. Gunderson described the BP as existing in a “dysphoric stalemate” in relationships, where intimacy needs are

unmet but where the requisite motivation and skills to assert the needs are non-existent. Gunderson's first stage resembled the "tension-building" phase of Walker's abuse cycle, during which frustrations increase. Gunderson's second stage occurred when the BP perceived an intimate relationship as possibly lost. Defensive behavior at this stage was expressed as anger, devaluation of the significant other, or open rage. This appeared to correspond to Walker's second "battering" phase of the abuse cycle. Gunderson's third stage occurred when the significant other was lost. At this point, the BP engaged in behaviors designed to ward off the subjective experience of aloneness. Impulsive substance use and promiscuity were the examples offered by Gunderson. Another example might be the exaggerated "appeasement" behaviors that assaultive husbands engage in after their wife has temporarily left the relationship. These behaviors persist until the woman has emotionally returned, when the cycle begins again.

In a series of studies, Dutton (1994a, b, 1995a, b, 1999, 2002) examined personality profiles of assaultive males to ascertain whether BPO, as described by Gunderson, was pronounced in this sample. The overall strategy of this work was based on self-report scales, filled out by court-referred abusive men as part of an assessment procedure for treatment, and used to establish correlations with their female partners' reports of their abusiveness. Both self-referred and court-referred men were compared to demographically matched controls (Dutton & Starzomski, 1993, 1994; Dutton 2006). Extensive analyses of the men's reporting tendencies were made via social desirability scales (Dutton & Hemphill, 1992). Self-reports of a man's anger, jealousy, experience of trauma symptoms, and abusiveness, and reports of his abusiveness (both physical and psychological) made by his female partner constituted the dependent variables in these studies. Self-reports were made on a scale measuring borderline personality organization (Oldham et al., 1985). The scale has three subscales: identity diffusion (a poorly integrated sense of self), primitive defenses (projection and splitting), and reality testing (transient psychotic states). In an initial sample of 80 wife assaulters and 40 demographically matched controls, Dutton and Starzomski (1994) found BPO scores to be similar to those for diagnosed borderlines. Furthermore, BPO scores were significantly related to chronic anger (measured using Siegel's Multidimensional Anger Inventory or MAI) (Siegel, 1986), jealousy, use of violence, and experience of adult trauma symptoms in the wife assault group. High-BPO scorers reported significantly more anger, of greater frequency, magnitude, and duration. Analysis of response styles indicated that these associations were not merely effects of disclosure or social desirability.

Dutton and Starzomski (1993) evaluated these findings by focusing on wives' reports of abusive treatment by their husbands through assessment of both physical and psychological abuse. Strong associations of men's BPO scores with women's reports of male abusiveness were found. A multiple regression indicated that BPO scale scores combined with scores from a self-report scale for anger (Siegel, 1986) accounted for 50% of women's reports of the husbands' use of dominance/isolation (PMWI Factor 1) and 35% of emotional abuse scores (PMWI Factor 2).

30.5 Conflict Studies

Heightened reactive anger (as distinct from chronic hostility; Eckhardt, Barbour, & Stuart 1997) is believed to increase the likelihood of adopting aggressive conflict resolution strategies (Konecni, 1975; Rule & Nesdale, 1976; Dodge, Petit, Bates, & Valente, 1995; Maiuro et al., 1988; Eckhardt, Barbour & Davidson, 1998; Jacobson, Gottman, Walz, et al., 1994, Chapter 14 by J.A. Hubbard et al., this book), while anxiety, on the other hand, is thought to be associated with the reduction or even the complete withdrawal of the use of such tactics (Schill & Schneide, 1970). However, in the studies reviewed here, anger and anxiety were correlated. In these studies, there was no possibility of an action component. It is possible that anger and anxiety are initially compounded and then become

distinct as a function of the action taken, with agency toward a target increasing anger and withdrawal increasing anxiety. Past relevant behavioral patterns may color present emotional reactions. Support for this notion comes from Dutton and Browning's (1988) finding that abusive males (abusiveness being viewed here as a tendency to use aggressive conflict resolution tactics) display heightened anger responses to scenarios of male–female conflict in comparison to non-abusive controls. This effect is especially pronounced when the conflict's theme touches on abandonment of the male. If elevated anger responses are observed among abusive males exposed to conflict, then it may be possible to use analogue testing (recording emotional reactions to taped conflicts) to identify who is likely to become angered and potentially abusive in real-life intimate conflicts.

Dutton and his colleagues (Dutton & Browning, 1988; Strachan & Dutton, 1992; Dutton et al., 1994; Jack et al., 1995) used audiotaped and videotaped simulated conflicts as an analogue to measuring individual reactions to real conflict. In these studies, an emotional “baseline” measurement was taken using the self-report Affect Adjective Checklist (AAC; Russell & Mehrabian, 1974, 1977). Subjects then observed or listened to an intense two person family conflict, following which they were asked to respond to a series of questions assessing their emotional reactions to and perceptions of the conflict (whose fault it was, whose side they took, etc.). Many of these conflicts involve parent–adolescent themes but some were couple focused, raising issues such as jealousy and potential abandonment of one partner by the other (Strachan & Dutton, 1992).

Strachan and Dutton (1992) had university students listen to jealousy-related conflicts and report affective responses using the Affect Adjective Checklist (Russell & Mehrabian, 1974) both prior to and following exposure to the conflict tapes. Before watching the conflict tapes, participants were put into either a low- or a high-power position with respect to the final edit of a group description of the conflict. Males and females showed significant post-exposure elevations in both anxiety and anger, with anger tending to be higher than any other affective reaction. Pre–post differences were examined for male-initiated and female-initiated conflicts. Women tended to report significantly more affect, with women's post-anger scores increasing by 114% above pre-exposure baseline compared to a 56% increase reported by the males. Power also significantly affected anger responses; individuals in the low-power condition tended to be angrier in response to witnessed conflict than individuals in the high-power condition.

The Strachan and Dutton (1992) study focused on jealousy, power, anger, and anxiety. Dutton et al. (1994) conducted a study that expanded on both the emotional measures taken and the range of conflict issues presented in previous studies. They examined anger, “subanger” (comprised of frustration, annoyance, and irritation), and anxiety reactions to conflict. Sixty undergraduates listened to conflict tapes of a variety of parent–teenager conflicts. Participants experienced low-anger and moderate anxiety in anticipation of the conflict exposure. As with the previous study, women's post-anger scores were disproportionately increased above pre-exposure baseline in comparison to the men's: 163% and 70%, respectively. Women also tended to show larger increases in the subanger composite than did men. However, when men's increases in anger were interpreted as a ratio of their overall increase in affect, these ratios were found to be greater than those calculated for the women. Anger scores in general were significantly greater than scores for any other measure of affect.

In both the Strachan and Dutton (1992) and the Dutton et al. (1994) study, anger reactions differed from anxiety reactions, with anger showing radical pre–post elevations, while anxiety was heightened only moderately. Jack et al. (1995) explored the relationship between previous exposure to specific parenting behaviors and anger in response to audiotaped family conflicts. The Jack et al. (1995) study used analogue conflict with an undergraduate sample to ascertain whether Strachan and Dutton's (1992) and Dutton et al.'s (1994) findings could be explained by the experience of abusive conflict resolution strategies learned in the family of origin. That is, the researchers wanted to assess

the extent to which emotional and behavioral responses of young adults could be explained by childhood experiences. During the prescreening portion of the experiment, participants were asked to answer a set of demographic questions and to fill out the Conflict Tactics Scale (CTS; Straus, 1979; Straus, Gelles, & Steinmetz, 1980). The CTS is a standardized scale used to evaluate the frequency and intensity with which subjects have used or experienced 19 conflict resolution tactics (including violence). To assess the use of such tactics in the family of origin, subjects rated the occurrence of these tactics in all family dyadic pairs (e.g., mother–you, father–you, father–mother). Respondents rated both their own and the interactants' use of these strategies. Two weeks later, subjects were again contacted at which time they completed the AAC both prior to and in response to the conflict tape exposure.

Heightened reactive anger levels prior to conflict exposure were associated with the experience of withdrawal or verbal abuse by parents during childhood and adolescence. Similarly, exposure to family of origin conflict characterized by withdrawal, verbal abuse, and physical abuse generated elevations in subanger (annoyance, frustration, and irritation) and total affect. Thus, it would appear that experience with specific family of origin conflict tactics might be related to elevated affective responding to anticipated conflict. The researchers suggest that greater anger and subanger in anticipation of and in response to conflict may be detrimental to an individual's conflict-solving ability. This conclusion is supported by the observation that physically abusive couples, prior to short-term problem-solving activities, presented with increased negative emotional state and arousal in comparison to non-abusive controls (Margolin, John, & Gleberman, 1989).

Dutton (1998) found that borderline traits in male abuse perpetrators were significantly correlated to their spouse's reports of abuse victimization. A resulting scale called the Propensity for Abusiveness Scale (PAS; Dutton 1995a, b) was developed that had good psychometric properties and was predictive of male abusiveness across a variety of samples. Thomas and Dutton (2004) used the PAS to predict emotional reactions of college students to exposure to conflict. Participants listened to taped conflict and filled out a battery of questionnaires. The PAS scores correlated strongly and significantly with two emotions that are counterproductive to conflict resolution: anger and anxiety. Pre–post conflict–exposure increases were observed for all affect measures, with the high PAS group (those individuals scoring in the fourth quartile) tending to be angrier and more anxious than the low PAS group. results suggested that PAS may be a reasonable predictor of affective reactions to conflict.

In both the Jack et al. (1995) study and the Thomas and Dutton (2004) study, participants showed an anticipatory effect to hearing the analogue conflict tapes. That is, after being told that they were going to hear the scenario involving intimate conflict, their level of anxiety and anger increased prior to actual exposure to the audiotapes. The participants' anger and anxiety levels increased still further after hearing the tapes.

30.6 The Articulated Thoughts in Simulated Situations Technique

In a series of studies Eckhardt, Barbour, & Davison, 1998; a technique called articulated thoughts in simulated situations (ATSS) has been refined and effectively used to capture cognitive distortions of persons exposed to interpersonal interactions. As with the research by Dutton and his colleagues described above, subjects listen to anger-arousing audiotapes which are stopped at crucial junctures at which time the subject provides open-ended reporting of his or her thoughts at that time (by verbalizing them into a tape recorder). These are then coded for cognitive distortions (illogical, faulty or misguided processing) or cognitive deficiencies using a coding manual developed to code for “an insufficient amount of cognitive activity in situations where more forethought would be

beneficial” (Eckhardt et al., 1998, p 261). Using this coding technique, Eckhardt et al. (1998) found that martially violent men articulated more irrational thoughts and cognitive biases during exposure than did non-martially violent men. These included the following cognitive biases associated with extreme anger: arbitrary inference – the making of assumptions or drawing conclusion in the absence of supporting evidence, selective abstraction – understanding an experience on the basis of one detail taken out of context while ignoring salient aspects of the situation, overgeneralization – constructing a general rule from one or a few isolated incidents and applying the rule generally, magnification – overestimating the incidence of events and reacting incongruously to the presenting situation, personalization – the tendency to engage in self-referent thinking when presented with situations having little to do with the self, dichotomous thinking – categorizing an event in one of two extremes, hostile attributions – blaming the cause of an event on malicious and hostile intentions of another. For an in-depth examination of these processes as they apply to intimate abusiveness, the reader is referred to Murphy and Eckhardt (2005).

30.7 Anger in Abusive Females

Besides the work on abusive females and “negative emotionality” (NEM) by Moffitt and Ehrensaft described above, Henning and his colleagues have found personality disturbance profiles in court-mandated female abusers that also have borderline features. Henning, Jones, and Holdford (2003) reported the demographic, childhood family functioning, and mental health characteristics for a large sample of male and female domestic violence offenders in Tennessee. They found few demographic differences between men and women arrested for domestic violence. Women were more likely to have attended college but were less likely to work outside the home. Analyses comparing childhood experiences (e.g., physical abuse, interparental physical aggression, parental criminal behavior, or substance abuse) that might result in adulthood adjustment difficulties or psychopathology revealed few gender differences. Men were more likely than women to report corporal punishment by primary caregivers and women were more likely to report witnessing severe abuse between their parents. Men and women were equally likely to report clinically significant distress. MCMI data for male and female perpetrators revealed that females were about five times more likely to have borderline peaks above 75 (considered to be the clinically significant cutoff point). In all, the Axis 2 personality disorder patterns found by Henning et al. indicated high levels of psychopathology in female offenders. The personality disorders observed (e.g., borderline PD) have anger as a central feature. This suggests that treatment focusing on personality disorders may be valuable for intimate abuse perpetrators of both sexes.

30.8 Interactive Anger Expression

Jacobson et al. (1994) recruited physically aggressive and martially distressed non-violent control couples to discuss “areas of disagreement” in a laboratory setting. Both martially violent husbands and wives displayed significantly more anger than did controls. Although the study focused on and reported profiles of abusive husbands, 50% of the wives committed severe acts of relationship abuse as well.

Margolin et al. (1989) and Burman, Margolin, and John (1993) started with an examination of interaction patterns in four different types of couples called physically abusive (PA), verbally abusive (VA), withdrawn but non-abusive (WI), and non-distressed and non-abusive (ND), based on

responses to the CTS and the Dyadic Adjustment Scale (Spanier 1976). In the early 1980s, self-report questionnaires were used to assess interaction style (questionnaires such as the "Communication Apprehension Inventory" or the Spouse Specific Assertiveness Scale); later these would be replaced by more sophisticated techniques for videotaping and scoring marital interaction.

Margolin noted that "the interactional processes of abuse and withdrawal seem to be related in some couples." Men in these groups showed particularly low self-disclosure. The physically abusive group also used withdrawal, as did the group that only withdrew but was not abusive. Margolin speculated that the physically aggressive group might use withdrawal as an initial response to conflict, then become abusive when the withdrawal failed. Perusal of her screening criteria reveals that these were bi-directionally violent couples (1984).

By 1988 Margolin and her colleagues had moved to assessment of in vivo interactions. Typically, couples would sign in for the research, undergo an initial screening/assessment, and then be asked to "discuss" two "problematic topics" (chosen from three offered in the screening self-reports of the couples). These discussions were videotaped for later coding. Experimenters observed the interaction through a one-way mirror and later reviewed the videotapes which were then coded (Margolin et al., 1989).

Coders assigned codes to the previous interaction into one of five summary categories: (1) offensive negative (mildly negative gestures, negative gestures, negative touch, and non-verbal command) (2) negative voice, (3) defensive negative (head hand, no eye contact, lean away, distract), (4) physical positive (positive gesture, positive touch), and (5) smile/laugh. Each discussion was independently coded by three coders. Obviously, before any conclusions can be drawn, the coders must agree with each other on their use of the coding system (exhibit inter-rater reliability). Depending on which code category was examined, inter-rater reliability ranged from 0.84 to 0.94, all highly acceptable. After each discussion, subjects reported their emotional reactions on four dimensions: sadness, anxiety, anger, and feeling attacked.

Margolin et al. (1989) found that the chief differentiating factor between the PA group and other groups was the husbands. PA husbands exhibited more instances of negative voice and more overtly negative behaviors than did husbands in other groups. PA husbands also reported more sadness, fear, anger, and feeling attacked (and somewhat more physiological arousal).

Despite the controlled and semi-public nature of the discussions, PA husbands exhibited negative affect patterns that were indicative of non-constructive approaches to conflict and could escalate into a more extreme expression of aggression. These included irate, angry, whining, yelling, sarcastic, nagging, lecturing, accusatory, mocking, and otherwise irritating voice tones. Negative behaviors included signs of dismissal, waving arms, pointing a finger at the other, threatening or mimicking gestures, and negative physical contact. The PA husbands tended not to exhibit head-hanging, no eye contact, or leaning away.

PA wives showed a greater escalation of offensive negative behaviors than did VA or WI wives during the middle portions of the discussion period and then showed a greater de-escalation in the final period.

Burman et al. (1993) instructed couples to recall a typical serious conflict and how it began, who said what to whom, how the conflict progressed, and how it ended. Couples then re-enacted these conflicts, and the re-enactments (which averaged 10 minutes in length) were videotaped and coded. This time the coding was based not on 15-s intervals but instead on "floor switches" (a statement of one person bounded on either side by a statement of the other). This created a series of "lags" from one person's action to the reaction of the other and so on. PA couples turned out to exhibit more hostile affect and more contingent behavior patterns involving anger. Non-distressed couples could "exit these negative interaction cycles quite quickly" (p. 37). The authors conclude that "contrary to images of women in abusive relationships as passive and reticent, the women in the types of PA

relationships presented here are angry with or contemptuous of husbands and are quick to respond to their husband's anger."

Hence, anger is central to intimate abusiveness and seems to present a residual reaction to early family of origin interactions. Anger reactions "prime" themselves in people who present certain psychological profiles predictive of abusiveness and this appears to occur in both genders.

References

- Becker, E. (1973). *The denial of death*. New York: Free Press.
- Bowlby, J. (1969). *Attachment and loss: Attachment*. New York: Basic Books.
- Bowlby, J. (1973). *Attachment and loss*. New York: Basic Books.
- Bowlby, J. (1977). The making and breaking of affectional bonds. *British Journal of Psychiatry*, 130, 201–210.
- Bowlby, J. (1980). *Attachment and loss*. New York: Basic.
- Burman, B., Margolin, G., & John, R. (1993). America's angriest home videos: Behavioral contingencies observed in home reenactments of marital conflict. *Journal of Consulting and Clinical Psychology*, 61(1), 28–39.
- Buss, A. H. & Durkee, A. (1957). An inventory for assessing different kinds of hostility. *Journal of Consulting Psychology*, 21, 343–349.
- Coleman, D. H. & Straus, M. A. (1992). Marital power, conflict and violence in a nationally representative sample of American couples. In M. A. Straus & R. J. Gelles (Eds.), *Physical violence in American families* (287–300). New Brunswick: Transaction.
- Dutton, D. G. (1994a). Behavioral and affective correlates of borderline personality organization in wife assaulters. *International Journal of Law and Psychiatry*, 17(3), 265–277.
- Dutton, D. G. (1994b). The origin and structure of the abusive personality. *Journal of Personality Disorder*, 8, 181–191.
- Dutton, D. G. (1995a). Male abusiveness in intimate relationships. *Clinical Psychology Review*, 15(6), 567–581.
- Dutton, D. G. (1995b). Trauma symptoms and PTSD-Like profiles in perpetrators of intimate abuse. *Journal of Traumatic Stress*, 8(2), 299–316.
- Dutton, D. G. (1998). *The abusive personality*. New York: Guilford Press.
- Dutton, D. G. (1999). The traumatic origins of intimate rage. *Aggression and Violent Behavior*, 4(4), 431–448.
- Dutton, D. G. (2002). *The Abusive Personality: Violence and control in intimate relationships*. New York: Guilford Press.
- Dutton, D. G. (2006). *The abusive personality* (2nd ed.). New York: Guilford.
- Dutton, D. G. & Browning, J. J. (1988). Power struggles and intimacy anxieties as causative factors of violence in intimate relationships. In G. Russell (Ed.), *Violence in intimate relationships*. Great Neck, New York: PMA Publishing.
- Dutton, D. G. & Hemphill, K. J. (1992). Patterns of socially desirable responding among perpetrators and victims of wife assault. *Violence and Victims*, 7, 29–39.
- Dutton, D. G. & Holtzworth-Munroe, A. (1997). The role of early trauma in males who assault their wives. In D. Cicchetti & R. Toth (Eds.), *The Rochester Symposium on Development* (379–402). NY: Rochester.
- Dutton, D. G., Saunders, K., Starzomski, A., & Bartholomew, K. (1994). Intimacy anger and insecure attachment as precursors of abuse in intimate relationships. *Journal of Applied Social Psychology*, 24(15), 1367–1386.
- Dutton, D. G. & Sonkin, D. J. (2003). *Intimate violence: Contemporary treatment innovations*. Binghamton, NY: Haworth Press.
- Dutton, D. G. & Starzomski, A. (1993). Borderline personality in perpetrators of psychological and physical abuse. *Violence and Victims*, 8(4), 327–337.
- Dutton, D. G. & Starzomski, A. (1994). Psychological differences between court-referred and self-referred wife assaulters. *Criminal Justice and Behavior: An International Journal*, 21(2), 203–222.
- Dutton, D. G., Webb, A. N., Ryan, L. (1994). Gender differences in anger/anxiety reactions to witnessing dyadic family conflict. *Canadian Journal of Behavioural Science*, 26(3), 353–364.
- Dodge, K. A., Petit, G. S., Bates, J. E., & Valente, E. (1995). Social information processing patterns that mediate the effect of early physical abuse on later conduct problems. *Journal of Abnormal Psychology*, 104(4), 632–643.
- Eckhardt, C., Barbour, K. A., & Davison, G. C. (1998). Articulated thoughts of maritally violent men during anger arousal. *Journal of Consulting and Clinical Psychology*, 66(2), 259–269.
- Ehrensaft, M. K., Cohen, P., Johnson, J. G. (2006). Development of personality disorder symptoms and the risk of partner violence. *Journal of Abnormal Psychology*, 115(3), 474–483.

- Ehrensaft, M. K., Moffitt, T. E., & Caspi, A. (2004). Clinically abusive relationships in an unselected birth cohort: Men's and women's participation and developmental antecedents. *Journal of Abnormal Psychology, 113*(2), 258–270.
- Follingstad, D. R., Bradley, R. G., Helff, C. M., & Laughlin, J. E. (2002). A model for predicting dating violence: Anxious attachment, angry temperament and need for relationship control. *Violence and Victims, 17*(1), 35–47.
- Gunderson, J. G. (1984). *Borderline personality disorder*. Washington, DC: American Psychiatric Press, Inc.
- Henning, K., Jones, A., & Holdford, R. (2003). Treatment needs of women arrested for domestic violence: A comparison with male offenders. *Journal of Interpersonal Violence, 18*(8), 839–856.
- Holtzworth-Munroe, A., Bates, L., Smutzler, N., Sandin, E. (1997). A brief review of the research on husband violence Part I: Maritally violent versus nonviolent men. *Aggression and Violent Behavior, 2*(1), 65–99.
- Jack, L. A., Dutton, D. G., Webb, A. N., & Ryan, L. (1995). Effects of early abuse on adult affective reactions to exposure to dyadic conflict. *Canadian Journal of Behavioural Science, 27*(4), 484–500.
- Jacobson, N., Gottman, J. M., Waltz, J., Babcock, J., & Holtzworth-Munroe, A. (1994). Affect, verbal content and psychophysiology in arguments of couples with a violent husband. *Journal of Clinical and Consulting Psychology, 62*(5), 962–988.
- Kernberg, O. (1977). The structural diagnosis of Borderline Personality Organization. In P. Hartocollis (Ed.), *Borderline personality disorders: the concept, the syndrome, the patient* (87–121). New York: International Universities Press.
- Koneckni, V. (1975). Annoyance, type and duration of postannoyance activity and aggression: The “cathartic effect”. *Journal of Experimental Psychology, 52*(1), 126–133.
- Maiuro, R. D., Cahn, T. S., Vitaliano, P. P., Wagner, B. C., & Zegree, J. B. (1988). Anger, hostility and depression in domestically violent versus generally assaultive men and nonviolent control subjects. *Journal of Consulting and Clinical Psychology, 56*, 17–23.
- Margolin, G., John, R. S., & Gleberman, L. (1989). Affective responses to conflictual discussions in violent and non-violent couples. *Journal of Consulting and Clinical Psychology, 56*(1), 24–33.
- Mauricio, A. M., Tein, J., & Lopez, F. G. (2007). Mediating influences of personality disorder characteristics between attachment and violence. *Violence and Victims, 22*(2), 139–157.
- Moffitt, T. E., Caspi, A., Rutter, M., & Silva, P. A. (2001). *Sex differences in antisocial behavior*. Cambridge: Cambridge University Press.
- Murphy, C. M. & Eckhardt, C. I. (2005). *Treating the abusive partner: An individualized cognitive-behavioral approach*. New York: Guilford.
- Oldham, J., Clarkin, J., Appelbaum, A., Carr, A., Kernberg, P., Lotterman, A., et al. (1985). A self-report instrument for Borderline Personality Organization. In T. H. McGlashan (Ed.), *The borderline: Current empirical research* (pp. 1–18). Washington, DC: American Psychiatric Press.
- Russell, J. A. & Mehrabian, A. (1974). Distinguishing anger and anxiety in terms of emotional response factors. *Journal of Consulting and Clinical Psychology, 42*, 79–83.
- Rule, B. G., & Nesdale, A. R. (1976). Evidence for a three-factor theory of emotions. *Journal of Research in Personality, 11*, 273–294.
- Schill, T., & Schneide, L. (1970). Guilt and self-report hostility. *Psychological Reports, 27*, 713–714.
- Siegel, J. M. (1986). The multidimensional anger inventory. *Journal of Personality and Social Psychology, 5*(1), 191–200.
- Spanier, G. (1976). Measuring dyadic adjustment: New scales for assessing the quality of marriage and similar dyads. *Journal of Marriage and the Family, 3*, 15–28.
- Strachan, C. E. & Dutton, D. G. (1992). The role of power and jealousy in anger responses to sexual jealousy. *Journal of Applied Social Psychology, 22*(22), 1721–1740.
- Straus, M. A. (1992). The conflict tactics scale and its critics: An evaluation and new data on validity reliability. In M. A. Straus and R. J. Gelles (Eds.), *Physical Violence in American Families*. New Brunswick, N.J.: Transaction Publishers.
- Straus, M. (1979). Measuring intrafamily conflict and violence: The Conflict Tactics Scales. *Journal of Marriage and the Family, 41*, 75–88.
- Straus, M. A., Gelles, R. J., & Steinmetz, S. (1980). *Behind closed doors: Violence in the American Family*. Garden City, NY: Anchor Doubleday Press.
- Thomas, L. & Dutton, D. G. (2004). The propensity for abusiveness scale (PAS) as a predictor of affective priming to anticipated intimate conflict. *Journal of Applied Social Psychology, 34*(1), 1–14.
- Tolman, R. M. (1989). The development of a measure of psychological maltreatment of women by their male partners. *Violence and Victims, 4*(3), 159–177.
- Walker, L. E. (1979). *The battered woman*. New York: Harper and Row.
- Westen, D. & Shedler, J. (1999). Revising and assessing Axis II. *American Journal of Psychiatry, 156*(2), 258–285.

Chapter 31

Don't Worry, Be Angry? Effects of Anger on Feelings, Thoughts, and Actions in Conflict and Negotiation

Gerben A. Van Kleef

Abstract This chapter reviews research on the role of anger in conflict and negotiation. I focus on three broad classes of dependent variables that I roughly call feelings, thoughts, and actions to refer to (1) affective states and interpersonal sentiments, (2) conscious thought processes, and (3) actual conflict behavior. In addition, I distinguish between intrapersonal and interpersonal effects of anger, with intrapersonal effects referring to the influence of an individual's anger on his or her own feelings, thoughts, and actions and interpersonal effects referring to the influence of one individual's anger on the feelings, thoughts, and actions of one or more other conflict parties. The review reveals that at the intrapersonal level, anger is associated with hostile feelings, biased perceptions and attributions, and competitive behavior. At the interpersonal level, anger sometimes elicits reciprocal hostility that motivates competition and sometimes strategic considerations that motivate cooperation. Recent studies that incorporated several moderators to reconcile these disparate sets of findings are discussed.

At all levels of society, social interactions frequently produce conflict. Irrespective of whether we focus on interactions between individuals, groups, organizations, or nations, conflicts are omnipresent. Conflicts occur when two or more parties have (or perceive) a divergence of interests (Pruitt & Carnevale, 1993). This divergence of interests may take on many guises, ranging from disagreement about the price of a second-hand car to marital disagreements or political disagreements, to name but a few. Conflicts may vary tremendously in terms of the stakes, the likelihood and possible consequences of stalemate, and the relationship between the parties. These vast differences notwithstanding, most – if not all – conflicts can be dealt with by means of negotiation, which may be defined as “a discussion between two or more parties with the apparent aim of resolving a divergence of interest and thus escaping social conflict” (Pruitt & Carnevale, 1993, p. 2). Often, the divergent interests that lie at the heart of conflict and negotiation give rise to intense emotions, which may in turn influence conflict development (Barry, Fulmer, & Van Kleef, 2004; Davidson & Greenhalgh, 1999). Of the range of emotions that may arise in conflict, anger is perhaps the most prominent and pervasive (Adler, Rosen, & Silverstein, 1998; Allred, 1999; Daly, 1991). For a thorough understanding of the dynamics of conflict resolution and escalation, it is therefore crucial to know how parties in conflict are influenced by their own and others' anger.

This chapter focuses on the consequences of anger in conflict and negotiation, with special emphasis on three classes of dependent variables that may be referred to roughly as feelings,

G.A. Van Kleef (✉)

Department of Social Psychology, University of Amsterdam, Amsterdam, The Netherlands
e-mail: g.a.vankleef@uva.nl

thoughts, and actions.¹ With *feelings* I refer to a broad class of phenomena encompassing affective states (e.g., moods and emotions) and interpersonal sentiments (e.g., impressions, liking). The term *thoughts* is used to refer to a variety of conscious cognitive activities that parties in conflict may exhibit, such as making attributions, drawing inferences, and developing a negotiation tactic. Finally, *actions* refer to the actual behaviors displayed by conflict parties, such as conceding, retaliating, or problem solving. Besides distinguishing among different types of dependent variables, it is useful to make a distinction between intrapersonal effects and interpersonal effects of anger (Morris & Keltner, 2000; Van Kleef, De Dreu, & Manstead, 2004a). *Intrapersonal* effects refer to the influence of an individual's anger on his or her own feelings, thoughts, and actions during conflict. *Interpersonal* effects refer to the influence of one individual's anger displays on the feelings, thoughts, and actions of one or more other conflict parties. Below I first review research on the intrapersonal effects of anger. Then I discuss more recent work on interpersonal effects. I conclude with some reflections on the state of the art and directions for future research.

31.1 Intrapersonal Effects

Most research on intrapersonal effects has focused on diffuse positive and/or negative affect (e.g., Carnevale & Isen, 1986; Baron, 1990; Baron, Fortin, Frei, Hauver, & Shack, 1990; Forgas, 1998). This research has recently been extensively reviewed elsewhere (see Barry et al., 2004). The present discussion will therefore be confined to studies that explicitly examined the intrapersonal effects of state anger as compared to one or more other discrete emotional states. In contrast to diffuse affect studies, discrete anger studies are surprisingly scarce. Below I review the most relevant findings of the few studies that have been conducted, distinguishing among effects on feelings, thoughts, and actions.

31.1.1 Feelings

The only empirical study I have been able to locate that directly speaks to the intrapersonal effects of discrete anger on feelings in conflict was performed by Allred, Mallozzi, Matsui, and Raia (1997), who investigated the effects of anger in a negotiation context. In a simulated negotiation experiment, they independently manipulated anger and compassion and found that negotiators with high levels of anger and low levels of compassion had less concern for the other's interests and had less desire to work with the other in the future than did negotiators who had more positive emotional regard for the other party. According to a theoretical analysis by Allred (1999), the low regard that angry negotiators have for each other may be part of the reason why anger often triggers aggression and retaliation.

Further insight into the workings of anger at the intrapersonal level is provided by research on justice and organizational behavior. Research has shown that perceived injustice is among the most powerful antecedents of anger (e.g., Barclay, Skarlicki, & Pugh, 2005; see also work on appraisal theory by Smith, Haynes, Lazarus, & Pope, 1993). The anger that is often produced by perceived injustice has been associated with feelings of hostility, aggression, and a desire for revenge and

¹ The antecedents of anger fall outside the scope of this chapter. For a comprehensive treatment of the causes of anger, the reader is referred to Chapters 11, 15, and 16 of this volume.

retaliation (Baron, Neuman, & Geddes, 1999; Kennedy, Homant, & Homant, 2004; Skarlicki & Folger, 1997). Together with the work by Allred (1999) and Allred et al. (1997) discussed above, these studies indicate that anger often triggers related negative sentiments that may contribute to a vicious cycle of negative feelings. As we shall see below, this negative spiral is further fueled by the attributions that people in conflict make.

31.1.2 Thoughts

In spite of a blossoming literature on affect and social cognition (see, e.g., Bower, 1981; Bower & Forgas, 2001; Forgas, 1995, 2001; Isen, 1999; Loewenstein, Weber, Hsee, & Welch, 2001; Schwarz & Clore, 1983), surprisingly few empirical studies have addressed the effects of discrete anger on conflict-related cognition. However, several lines of research provide useful indirect evidence. For example, Lerner and Keltner (2001) demonstrated that anger influences individuals' risk perceptions and preferences. Specifically, angry people reported more optimistic risk estimates than did fearful people, suggesting that anger may reduce barriers that otherwise prevent individuals from exhibiting risky behavior. To the extent that competitive negotiation behavior (e.g., tough demands, hostile communication) may be seen as risky (Pruitt, 1981; Van Kleef, Steinel, van Knippenberg, Hogg, & Svensson, 2007), it is conceivable that anger similarly reduces individuals' reluctance to engage in competition.

Further suggestive evidence for the possible effects of anger on conflict parties' thinking is provided by a study on negative affect in negotiation by Forgas (1998). Forgas used a false-feedback technique to manipulate participants' emotional state prior to the negotiation. Participants who were led to believe that they had performed poorly planned and reported more competitive bargaining strategies than did those who received no feedback. To the extent that the experimental procedure employed in this study elicited discrete anger (as opposed to a diffuse negative mood), this finding tells us something about the possible intrapersonal effects of anger on conflict-related thoughts. Ostensibly, feelings of anger trigger a competitive mindset that may predispose individuals to engage in contentious behavior.

Anger also affects social perception. For instance, research has shown that, relative to sad or neutral participants, angry participants are slower to associate positive (as opposed to negative) traits with members of an outgroup (DeSteno, Dasgupta, Bartlett, & Caidric, 2004). Other research indicates that angry individuals exhibit lower trust (Dunn & Schweitzer, 2005). Furthermore, anger has been shown to promote heuristic (shallower) information processing, leading individuals to rely more on superficial cues and to pay less attention to the quality of arguments (Bodenhausen, Sheppard, & Kramer, 1994; Chapter 18 by D. Schultz et al., this volume; Tiedens & Linton, 2001; see Chapter 17 by P.M. Litvak et al., this book; Lerner & Tiedens, 2006, for a comprehensive review of the effects of anger on individual cognition). By the same token, several studies indicate that anger influences the way in which people in conflict interpret one another's behavior. In this regard, Allred (1999) discusses the implications of attribution theory (Heider, 1958; Kelley, 1967; Weiner, 1995) for our understanding of the role of anger in conflict. According to Allred's analysis, angry conflicts often stem from biased attributions of behavior.

To the extent that anger affects individuals' perceptions of their social worlds (e.g., Keltner, Ellsworth, & Edwards, 1993), it may increase the likelihood that negotiators fall prey to the *fundamental attribution error*, that is, the tendency to over-attribute other people's behavior to dispositions (Jones & Davis, 1965; Jones & Harris, 1967; Ross, 1977) and their own behavior to the circumstances (i.e., the *actor-observer bias*; Jones & Nisbett, 1971). Indeed, one of the "core relational

themes” of anger is other-blame (Roseman, Antoniou, & Jose, 1996; Smith & Lazarus, 1993). Research indicates that anger enhances the tendency to blame others for negative events (Quigley & Tedeschi, 1996), thus creating a recursive loop – the more anger one feels, the more one perceives others as responsible for a negative event, and vice versa (Lerner & Tiedens, 2006). This type of attributive pattern may lead an angry negotiator to blame his or her opponent even more for the adverse situation both are in, while washing his or her own hands of responsibility. Combined with the competitive thoughts that are ignited by feelings of anger, such attributions have strong potential to arouse a vicious cycle of hostile acts and counteracts, which may be reinforced by the negative interpersonal sentiments that anger also triggers. An obvious yet important question that arises is whether this explosive cocktail of hostile feelings, biased perceptions, and competitive thoughts actually fuels competitive behavior. As we shall see below, several studies suggest that it does.

31.1.3 Actions

In contrast to the rather embryonic state of knowledge of the intrapersonal effects of anger on feelings and thoughts in conflict, ample research speaks to the effects of anger on observable conflict behavior. In an early study, Baron et al. (1990) had subjects negotiate with an accomplice who disagreed with their point of view on a particular topic, manipulating the manner in which this disagreement was expressed. In half the conditions the accomplice expressed disagreement in a calm, reasonable, and non-provocative manner (e.g., “I can see why you feel that way, but I guess I disagree. . .”). In the other conditions, disagreement was expressed in an arrogant, condescending, and provoking fashion (e.g., “Oh come on, you’ve got to be kidding!”). Previous research (Baron, 1984) had shown that this procedure induces negative emotions, including anger. Baron et al.’s results revealed that male (but not female) participants who had been provoked prior to the negotiation made significantly less favorable initial offers to the accomplice than did those who had not been provoked.

Comparable findings were obtained in a more recent study on emotions in dyadic negotiation reported by Butt, Choi, and Jaeger (2005). They measured both negotiating parties’ levels of anger, pride achievement, gratitude, and guilt–shame. Among other effects they found a positive and significant association between anger and dominating behavior, indicating that negotiators with higher levels of anger adopted a more competitive stance in the course of the negotiation than did those with lower levels of anger. In addition, the study by Allred et al. (1997) mentioned above showed that negotiators who felt high anger and low compassion for each other achieved lower joint gains, presumably in part because the anger reduced their creative problem-solving abilities.

Along similar lines, Knapp and Clark (1991) examined the effects of anger and a number of other emotions in a resource dilemma, a situation in which individual interests are at odds with collective interests. In a laboratory simulation, participants harvested fish from a common and depletable resource pool that was only partially replenished at fixed time intervals. In this situation, the dilemma consists in the fact that although it is profitable for individual fishers to maximize their selfish interests by harvesting all they can, if all fishers were to do so the resource would be depleted and everyone would be collectively worse off (for overviews of research on these and other social dilemmas, see Dawes, 1980; Messick & Brewer, 1983). Before playing this fishing dilemma game, participants were experimentally induced to feel angry, sad, or happy (or neutral in a control condition). Across two experiments, Knapp and Clark found that angry and sad participants were more competitive (i.e., they took more fish) than were participants in a happy or neutral state.

Finally, in what has become a classic study, Pillutla and Murnighan (1996) examined the intrapersonal effects of unfairness and anger on the rejection of low ultimatum offers. In ultimatum bargaining, two players have to decide on how to distribute a certain amount of money or points. One of the players offers a proportion of the money to the other player, the recipient. If the recipient accepts, the money is distributed as proposed (Güth, Schmittberger, & Schwarze, 1982). Pillutla and Murnighan manipulated the extent to which recipients could evaluate the fairness of the proposer's offer by providing either complete or incomplete information about the amount of money that was to be divided. They found that participants were more likely to reject an offer if they were able to assess its unfairness and, more important, that this effect could be explained by increased levels of anger.

31.1.4 Recap and Remarks

One thing that becomes clear from this review is that most research on the intrapersonal effects of anger has focused on behavioral effects. Few studies directly speak to the (mutually related) effects of anger on feelings and thoughts. This bias reflects an understandable tendency of researchers to try and predict the effects of anger on readily observable variables such as the demands people make and the way resources are eventually distributed. Although a focus on behavioral effects is certainly praiseworthy, the downside of this approach is that the current body of empirical work does not provide much insight into *why* and *how* anger produces these behavioral effects. One possible explanation is suggested by Allred et al.'s (1997) finding that angry conflict parties show less concern for one another. Perhaps the reduced concern for others lowers individuals' reluctance to be purely egoistic. Another possibility is suggested by research on emotions and risk taking. It may be that anger "clouds" perceptions of danger and disposes individuals to take more risks (Lerner & Keltner, 2001), which could become manifest in (risky) competitive behavior. Yet another explanation can be derived from the mood management literature, which has documented that individuals in a negative emotional state are often motivated to take action to improve their mood (Mayer, Salovey, Gombert-Kaufman, & Blainey, 1991). Following this reasoning, an angry negotiator may be motivated to improve his or her emotional state by trying to get the most out of the negotiation. Clearly, future research is needed to provide more insight into the mechanisms underlying the intrapersonal effects of anger on conflict behavior.

Despite the unbalanced state of empirical affairs, the general pattern that emerges from the sparse data that are currently available is remarkably coherent. Across the board, the experience of anger has consistent effects on feelings, thoughts, and actions in conflict. Angry individuals tend to show less concern for their partner's welfare, have less desire to interact with the same partner in the future, feel hostility, experience retaliatory desires, perceive less risk, attribute their partner's (competitive) behavior to internal rather than external causes, plan more competitive bargaining strategies, and exhibit more self-centered behavior and contentious conflict strategies than do non-angry individuals. Arguably, these effects mutually reinforce one another. That is, biased perceptions of one's counterpart's intentions may exacerbate negative sentiment and worsen the interpersonal climate, and vice versa. Furthermore, both processes pave the way for selfish, competitive behavior that in turn feeds back into negative perceptions, biased attributions, and a poor climate. If we adopt a relational perspective, the available evidence thus paints a rather grim picture of anger – it biases social judgment, triggers a competitive mindset, and stimulates selfish behavior. But how do these effects play out at the interpersonal level? Does anger indeed merely disturb interpersonal relations or does it also have beneficial effects? This question is addressed in the next section, which examines how expressions of anger affect interpersonal dynamics.

31.2 Interpersonal Effects

Emotions, in general, and anger, in particular, are not just an individual state of the mind. They also have important interpersonal implications. In this regard, Parkinson (1996) argued that emotions should be primarily conceptualized as social rather than individual phenomena. Indeed, inspired by the early writings of Darwin (1872), researchers have identified a number of crucial social functions of emotions (e.g., Frijda, 1986; Frijda & Mesquita, 1994; Keltner & Haidt, 1999; Oatley & Jenkins, 1992). At the interpersonal level, emotions convey information to others about an individual's feelings (Ekman, 1993), social intentions (Fridlund, 1992; Van Kleef et al., 2004a), and orientation toward the relationship (Knutson, 1996). Further, emotions may evoke reciprocal or complementary emotions in others that may in turn help individuals respond to significant social events (Keltner & Haidt, 1999). And lastly, emotions serve as incentives or deterrents for others' social behavior (Klannert, Campos, Sorce, Emde, & Svejda, 1983). In light of these social functions, it is unsurprising that anger has significant interpersonal consequences. In this section I review the growing body of research on the interpersonal effects of anger in conflict and negotiation, again distinguishing among effects on feelings, thoughts, and actions.

31.2.1 *Feelings*

As one might expect, research has documented pervasive interpersonal effects of anger expressions on observers' own emotions and on interpersonal liking. In keeping with more general theorizing and research on emotional contagion (see, e.g., Hatfield, Cacioppo, & Rapson, 1994), studies on the interpersonal effects of emotions in conflict and negotiation have documented that anger often spreads from one negotiator to the other. For example, Van Kleef et al. (2004a) demonstrated that negotiators who are confronted with an angry opponent tend to become angry themselves. Likewise, Friedman et al. (2004) found, in study of anger in online mediation, that disputants' anger expressions elicited anger in their partners.

In a related vein, negotiators dealing with an angry (as opposed to a happy or non-emotional) opponent have been shown to develop a more negative impression of the other (Van Kleef et al., 2004a), to be less satisfied with the negotiation afterward, and to be less willing to engage in future interaction with the same party (Kopelman, Rosette, & Thompson, 2006; Van Kleef, De Dreu, & Manstead, 2004b). Apparently, just as at the intrapersonal level, anger reduces the quality of the interpersonal climate. At the intrapersonal level it does so by negatively affecting the angry negotiator's regard for his or her counterpart. At the interpersonal level, one negotiator's expressions of anger negatively affect the sentiments of observers. Together, these two processes thus make for an especially unconstructive atmosphere.

31.2.2 *Thoughts*

A couple of studies have addressed the interpersonal effects of anger on negotiators' thoughts and judgments. In an early study, Van Kleef et al. (2004a) investigated the interpersonal effects of anger and happiness in a computer-mediated negotiation simulation. Among other things, they found that negotiators used their opponent's emotions to track his or her limit, that is, the least favorable negotiation outcome they would be willing to accept. Specifically, negotiators who were confronted with an angry opponent estimated the opponent's limit to be high, whereas negotiators with a happy

opponent judged the opponent's limit to be low. Compatible findings were obtained by Sinaceur and Tiedens (2006). In a scenario study and in a face-to-face negotiation experiment in which one of the negotiators was instructed to display either anger or no emotion, they demonstrated that angry opponents were seen as tougher than non-emotional ones.

These studies indicate that although at the intrapersonal level of analysis the effects of anger on thoughts tend to parallel the effects on feelings (e.g., anger triggers both hostile feelings and a competitive mindset), at the interpersonal level the effects of anger on feelings and thoughts are qualitatively different. As we have seen, negotiators who are confronted with an angry counterpart develop negative feelings and impressions of their counterpart. Combined with the consistent link between anger and competitive tendencies and behavior uncovered by research on the intrapersonal effects of anger, this suggests that expressions of anger elicit competitive behavior in observers. However, we have also seen that expressions of anger may trigger strategic inferences in observers that may actually motivate cooperative behavior. Research on "mismatching" suggests that negotiators become more cooperative to the extent that they perceive their partner as tough and intransigent, because they are motivated to avoid an unprofitable impasse (e.g., Pruitt, 1981; Van Kleef et al., 2004a). Such considerations can thus be expected to make negotiators respond in a conciliatory manner to their opponent's anger expressions.

31.2.3 Actions

In contrast to the consistent relation among feelings, thoughts, and actions in response to anger at the intrapersonal level, these relationships are rather evasive at the interpersonal level. According to the analysis presented above, negotiators' feelings and thoughts would seem to push them in opposing behavioral directions. The question thus arises as to which of the two is more predictive of behavior. As we shall see, there is evidence for both. Below I first discuss evidence for the link between feelings and actions in response to anger. Then I review evidence for the relationship between thoughts and actions. Finally, I discuss recent research that has uncovered moderator variables that push the balance in one or the other direction.

31.2.3.1 From Feeling to Acting

We have seen that expressions of anger generally elicit negative emotions and impressions in observers, suggesting that expressions of anger are detrimental to a negotiator's outcomes. Although this may seem like an obvious idea, there is surprisingly little direct evidence to support it. One of the few studies that directly examined the role of observers' feelings in determining their responses to others' expressions of anger was conducted by Friedman et al. (2004). They used data from a company that offers online mediation services for people who conduct business through the Internet. They found that attempts at conflict resolution were more likely to break down when one of the disputants expressed anger at the other, except when the recipient of the anger had a weak negotiation position that constrained his or her negotiation latitude. Importantly, Friedman and colleagues also found that the harmful effects of anger expressions were in part mediated by the negative emotional responses they generated in observers.

Complementary evidence is reported by Kopelman et al. (2006), who examined the effects of various emotional strategies in a dispute simulation, an ultimatum setting, and a distributive negotiation (a situation in which one party's gain equals the other's loss and there are no opportunities for a "win-win" solution). Across three studies, they found that negotiators who strategically displayed

negative emotions (possibly including discrete anger) were less likely to close a deal, extract concessions, and incorporate a future business relationship in the contract than were those who strategically displayed positive or neutral emotions. Thus, in support of a *feeling-to-action* hypothesis, these studies indicate that expressions of anger may indeed produce unfavorable outcomes for the expresser and that these effects are due at least in part to the elicitation of negative emotions such as anger in observers.

31.2.3.2 From Thinking to Acting

Several studies speak to the role of negotiators' conscious thinking in determining their responses to their counterparts' emotional displays. Van Kleef et al. (2004a) investigated the interpersonal effects of anger and happiness in negotiations. Over the course of a computer-mediated negotiation, participants received information about their (simulated) opponent's emotional state. For example, participants read messages from their opponent saying that "this offer makes me really angry" or "this offer makes me really happy." The results showed that participants with an angry opponent made larger concessions than did participants with a non-emotional opponent, whereas participants with a happy opponent made smaller concessions. Importantly, this effect was fully mediated by the focal negotiator's appraisal of the opponent's limit or "rock bottom." Negotiators who were confronted with an angry (as opposed to a happy) opponent estimated the opponent's limit to be higher, leading them to concede to avoid an unfavorable impasse. Likewise, Sinaceur and Tiedens (2006) found that angry negotiators extracted larger concessions from their opponents because they appeared tougher than non-emotional negotiators.

31.2.3.3 Moderators

These studies suggest that the interpersonal effects of anger are the result of a process of strategic decision making on the part of the emotion-perceiving negotiator. Negotiators use the information about the other's emotion to inform their own negotiation strategy. This inferential process requires a certain amount of cognitive motivation and ability. Accordingly, Van Kleef et al. (2004b) found support for a moderating role of epistemic motivation – the motivation to engage in systematic information processing to develop a thorough understanding of the situation (Kruglanski, 1989). In two computer-mediated negotiation experiments and a field study among managers of various companies, they found that the interpersonal effects of anger were moderated by the need for cognitive closure (a personality characteristic that influences information processing tendencies; Kruglanski & Webster, 1996; Webster & Kruglanski, 1994), time pressure (a situational determinant of information processing tendencies; De Dreu, 2003), and number of alternatives to a negotiated agreement (a situational determinant of power and concomitant information processing depth; De Dreu & Van Kleef, 2004; Fiske & Dépret, 1996; Keltner & Robinson, 1997; Pinkley, 1995). Participants with a low need for cognitive closure, under low time pressure, or with low power (i.e., high epistemic motivation) were strongly affected by the opponent's emotion, leading them to concede more to an angry opponent than to a non-emotional or happy one. In contrast, those with a high need for closure, under high time pressure, or with high power (i.e., low epistemic motivation) were unaffected by the opponent's emotion. Additional analyses revealed that the moderating effect of time pressure was mediated by information processing – participants who negotiated under high time pressure engaged in less thorough information processing, which rendered them impervious to the strategic implications of their counterpart's anger.

In their online mediation study, Friedman et al. (2004) obtained compatible findings. They reasoned that responses to another's anger expressions depend on one's position in the negotiation.

Negotiators who are in a weak position, they argue, are more likely to respond in a conciliatory fashion to an opponent's anger than are negotiators occupying a strong position. In line with this reasoning, Friedman et al. found that expressions of anger elicited concessions when observers had a weak position (i.e., an unfavorable reputation) but not when they had a strong position (i.e., a favorable reputation). Likewise, Sinaceur and Tiedens (2006) found that only negotiators who had few alternatives to a negotiated agreement (i.e., a weak negotiation position) conceded more to an angry opponent than to a neutral one. Finally, Van Kleef, De Dreu, Pietroni, and Manstead (2006) reported converging evidence for a moderating role of power based on different samples (students, general population, managers), research methods (experiment, field simulation, scenarios), and power bases (number of alternatives, quality of best alternative, managerial support, and legitimate power). In a series of five studies, they showed that negotiators with few or poor alternatives to a negotiated agreement, little support from their management, or low legitimate power (power based on one's position in an organization) were strongly affected by their opponent's anger, whereas those with many or highly attractive alternatives, strong support from management, or high legitimate power were immune to the opponent's anger. Thus, in support of a *thought-to-action hypothesis*, these studies suggest that responses to anger in conflict are often motivated by strategic considerations rather than by affective tendencies.

31.2.3.4 Integrating Feelings, Thoughts, and Actions

Some of the studies described above provide support for feeling-based reactions to expressions of anger, whereas other studies support the idea that responses to displays of anger are mediated by strategic thinking. Although some of these studies have identified moderators that strengthen or weaken these effects (i.e., Friedman et al., 2004; Sinaceur & Tiedens, 2006; Van Kleef et al., 2004b, 2006), none of them has been able to provide an integrated account of both beneficial and detrimental effects of anger by incorporating both feeling-based and thought-based processes in a single study. Below I discuss two recent studies that offer such an integrative approach.

Inspired by the classic advice to "separate the people from the problem" (Fisher & Ury, 1981), Steinel, Van Kleef, & Harinck (2008) differentiated between emotions that are directed toward a negotiator's *offer* and emotions that are directed toward the negotiator *as a person* to reconcile the seemingly contradictory findings concerning the effects of anger on negotiator concessions. They reasoned that expressing anger about the situation will lead an opponent to make large concessions, as can be predicted on the basis of a strategic decision-making perspective (see Van Kleef et al., 2004a, b). On the other hand, they contend, directing anger toward the person may produce a quite different effect. When the anger is directed toward the person instead of their offer, in a sense it loses some of its informative qualities. When anger is explicitly directed toward one's offer, it is relatively clear what the behavioral implications are – one should make a better offer. However, when the anger is directed toward oneself as a person, it is less clear how one should respond. Furthermore, expressing anger at someone personally may elicit strong feelings of anger and antipathy in that person, which may in turn cause them to take a more competitive stance (see Friedman et al., 2004; Kopelman et al., 2006).

To test the validity of this reasoning, Steinel et al. (2008) had students engage in a computer-mediated negotiation against a pre-programmed opponent who expressed either anger or happiness by means of verbal messages (cf. Van Kleef et al., 2004a, b). The anger (or happiness) was either person directed (e.g., "this offer makes me really angry") or behavior directed (e.g., "this person makes me really angry"). The results supported the above reasoning. When emotional statements were directed at the participant's offer, participants used the opponent's emotion to assess his or her limits and, accordingly, they conceded more to an angry opponent than to a happy one. However,

when the emotions were directed at the negotiator as a person, negotiators conceded *less* to an angry opponent than to a happy one. In this case, participants did not see useful information in their opponent's emotions, and accordingly there was no mediation of perceived limits. As a result, negotiators reacted more competitively to angry rather than happy counterparts by standing firm and making small concessions.

Van Kleef and Côté (2007) integrate the feeling-based and thought-based perspectives in a slightly different, yet related, way. They propose a dual-process model in which the appropriateness of the anger and the relative power jointly determine whether a negotiator will respond to an opponent's anger by retaliating and placing competitive demands or by accommodating and conceding value. The central tenet of the model is that an opponent's expressions of anger may elicit two distinct processes. On the one hand, anger may elicit reciprocal anger and retaliatory behavior via an emotional route (cf. Friedman et al., 2004; Kopelman et al., 2006). On the other hand, anger may signal toughness and unwillingness to concede, which may elicit concessions in observers via a strategic route (cf. Sinaceur & Tiedens, 2006; Van Kleef et al., 2004a, b). The model posits that the relative predictive power of these two processes depends on the power of the observing negotiator and the perceived appropriateness of the anger.

In two experiments using different methods and operationalizations of power and appropriateness of anger, Van Kleef and Côté (2007) obtained support for the dual-process model. First of all, results indicated that inappropriate expressions of anger (e.g., expressions that violated a display rule; cf. Matsumoto, 1993; Rafaeli & Sutton, 1987) created a stronger desire to retaliate than did appropriate expressions of anger. Second, the appropriateness of the opponent's anger did not affect the behavior of low-power negotiators – they conceded uniformly to both appropriately angry and inappropriately angry opponents (relative to non-emotional counterparts). In contrast, the appropriateness of the counterpart's anger did matter when the focal negotiator had high power. High-power participants, besides conceding less in general, were especially intransigent when the opponent expressed inappropriate anger. Thus, anger may backfire if it is directed at a powerful opponent who perceives the anger as inappropriate.

31.2.4 Recap and Remarks

A growing body of research speaks to the interpersonal effects of anger on feelings, thoughts, and actions in conflict. The conclusions emerging from this research are quite consistent when it comes to feelings and thoughts. With regard to feelings, converging evidence shows that expressions of anger tend to arouse negative emotions and impressions in observers. As to thoughts, several studies indicate that expressions of anger may trigger strategic inferences in observers, including appraisals of the expresser's limits and toughness. We have also seen that negotiators are more likely to make such inferences when they have high epistemic motivation.

Findings pertaining to the interpersonal effects of anger on actual conflict behavior are more equivocal. Some studies indicate that expressions of anger elicit cooperation, whereas others indicate that anger triggers competition. Interestingly, the underlying mechanisms that have been uncovered in some of these studies map quite nicely onto the feelings versus thoughts distinction. The link between expressions of anger and cooperation, on the one hand, is consistently mediated by strategic considerations relating to perceptions of the opponent's toughness and limits. The link between expressions of anger and competition, on the other hand, appears to be at least partially mediated by reciprocal anger. In other words, it seems that responses to expressions of anger in conflict and negotiation may be motivated by feeling-based processes on the one hand and thought-based processes on the other hand (see Van Kleef, 2009 for a more elaborate discussion of this idea). Indeed,

recent evidence indicates that both processes may predict the interpersonal effects of anger on behavior depending on factors such as the target of the emotion and the appropriateness of the anger expression.

31.3 Conclusion

Overall, the current state of the art regarding the role of anger in conflict paints a somewhat grim picture. At the intrapersonal level, anger triggers hostile feelings, biased perceptions and attributions, and competitive behavior. At the interpersonal level, expressions of anger elicit negative impressions, reciprocal hostility, and sometimes retaliation. Moreover, some of these effects appear to be mutually enhancing. Biased perceptions of one's counterpart's intentions may exacerbate negative sentiment and further spoil the interpersonal climate, and vice versa. Furthermore, both processes pave the way for competitive, retaliatory behavior that in turn fuels negative perceptions, faulty attributions, and a bad interpersonal relationship. Finally, effects at both levels of analysis may work in concert to get in the way of constructive problem solving and mutually satisfying agreement. For example, one party's anger may lead him or her to respond especially aggressively to the other's anger displays of anger, thereby increasing chances of hurting impasse and a damaged relationship.

On a more positive note, anger also appears to have some beneficial effects. The generally negative effects of anger summarized above notwithstanding, anger can be thought of as a functional in that it communicates a sincere commitment to a particular position or course of action (Daly, 1991; Putnam, 1994). For instance, the economist Robert Frank argued in his influential book *Passions within reason: The strategic role of the emotions* that "irrational" anger often pays. As Frank (1988) points out, it might be considered irrational to hit somebody who parks in one's parking space, given the likelihood of prosecution. However, if one becomes known as the type of person who easily loses his/her temper, it is likely that the neighbors will keep one's parking space free. Some of the effects reviewed in this chapter – especially the finding that anger can elicit concessions by signaling toughness and high limits (Sinaceur & Tiedens, 2006; Van Kleef et al., 2004a; Chapter 21 by D.M.T. Fessler, this book) – resonate nicely with this idea.

Expressing anger may also entail less cynical benefits. Averill (1982) found that people recalling experiences in which they got angry evaluated the majority of the consequences as beneficial, for example, because their anger led the object of the anger to change his or her attitude or behavior. Along similar lines, Fischer and Roseman (2007) argue that the social function of anger is to obtain a better outcome by forcing a change in another person's behavior. They found that anger is often characterized by short-term attack responses but long-term reconciliation. Their analysis suggests that expressing anger may help one to enforce changes in another's behavior without necessarily precluding a good relationship. It should be noted, however, that Fischer and Roseman's data also suggest that the constructive long-term effects of anger are more likely in intimate relationships. Most of the research reviewed in this chapter concerns conflicts in exchange relationships that are characterized by relatively low levels of intimacy, which could account for the generally negative interpersonal effects of anger on relationship quality observed in these studies. Clearly, future research is needed to shed more light on both the short-term and the long-term effects of anger in conflict in both communal and exchange relationships.

Another avenue for future investigation – and one that is as yet largely unpaved – concerns the interaction between the intra- and the interpersonal effects of anger over time. It is likely that effects at both levels of analysis mutually reinforce one another, such that one party's anger affects not only their own feelings, thoughts, and actions but also those of the other party, and vice versa. In fact, such recursive processes may well be among the core causes of intractable conflict. Incorporating a

combination of controlled laboratory experiments and more naturalistic, possibly longitudinal studies seems to be a promising strategy to uncover how these complicated patterns of mutual influence develop over time.

Finally, future studies are needed to gain more insight into the relations among anger, feelings, thoughts, and actions in conflict, especially in the interpersonal domain. Better insight into when, how, and why behavior in conflict is predicted by affective reactions versus more deliberate, strategic responses to other's anger is crucial if we are to truly understand the role of anger in conflict. It is my hope that this chapter will stimulate future research endeavors so that this intriguing and important area of inquiry can continue to blossom for years to come – hopefully leading, eventually, to a true understanding of the role of anger in conflict.

Acknowledgments Preparation of this chapter was facilitated by a research grant from the Netherlands Organisation for Scientific Research (NWO-VENI 451–05-010). I thank Astrid Homan for her comments on an earlier draft of this chapter.

References

- Adler, R. S., Rosen, B., & Silverstein, E. M. (1998). Emotions in negotiation: How to manage fear and anger. *Negotiation Journal*, 14, 161–180.
- Allred, K. G. (1999). Anger and retaliation: Toward an understanding of impassioned conflict in organizations. *Research on Negotiation in Organizations*, 7, 27–58.
- Allred, K. G., Mallozzi, J. S., Matsui, F., & Raia, C. P. (1997). The influence of anger and compassion on negotiation performance. *Organizational Behavior and Human Decision Processes*, 70, 175–187.
- Averill, J. R. (1982). *Anger and aggression*. New York: Springer.
- Barclay, L. J., Skarlicki, D. P., & Pugh, S. D. (2005). Exploring the role of emotions in injustice perceptions and retaliation. *Journal of Applied Psychology*, 90, 629–643.
- Baron, R. A. (1984). Reducing organizational conflict: An incompatible response approach. *Journal of Applied Psychology*, 69, 272–279.
- Baron, R. A. (1990). Environmentally induced positive affect: Its impact on self-efficacy, task performance, negotiation, and conflict. *Journal of Applied Social Psychology*, 20, 368–384.
- Baron, R. A., Fortin, S. P., Frei, R. L., Hauver, L. A., & Shack, M. L. (1990). Reducing organizational conflict: The role of socially-induced positive affect. *International Journal of Conflict Management*, 1, 133–152.
- Baron, R. A., Neuman, J. H., & Geddes, D. (1999). Social and personal determinants of workplace aggression: Evidence for the impact of perceived injustice and the type A behavior pattern. *Aggressive Behavior*, 25, 281–296.
- Barry, B., Fulmer, I. S., & Van Kleef, G. A. (2004). I laughed, I cried, I settled: The role of emotion in negotiation. In M. J. Gelfand & J. M. Brett (Eds.), *The handbook of negotiation and culture* (pp. 71–94). Palo Alto, CA: Stanford University Press.
- Bodenhausen, G. V., Sheppard, L. A., & Kramer, G. P. (1994). Negative affect and social judgment: The differential impact of anger and sadness. *European Journal of Social Psychology*, 24, 45–62.
- Bower, G. H. (1981). Mood and memory. *American Psychologist*, 36, 129–148.
- Bower, G. H. & Forgas, J. P. (2001). Mood and social memory. In J. P. Forgas (Ed.), *Handbook of affect and social cognition* (pp. 95–120). Mahwah, NJ: Erlbaum.
- Butt, A. N., Choi, J. N., & Jaeger, A. M. (2005). The effects of self-emotion, counterpart emotion, and counterpart behavior on negotiator behavior: A comparison of individual-level and dyad-level dynamics. *Journal of Organizational Behavior*, 26, 681–704.
- Carnevale, P. J. & Isen, A. M. (1986). The influence of positive affect and visual access on the discovery of integrative solutions in bilateral negotiation. *Organizational Behavior and Human Decision Processes*, 37, 1–13.
- Daly, J. P. (1991). The effects of anger on negotiations over mergers and acquisitions. *Negotiation Journal*, 7, 31–39.
- Darwin, C. (1872). *The expression of the emotions in man and animals* (3rd ed.). London: HarperCollins.
- Davidson, M. N. & Greenhalgh, L. (1999). The role of emotion in negotiation: The impact of anger and race. *Research on Negotiation in Organizations*, 7, 3–26.
- Dawes, R. M. (1980). Social dilemmas. *Annual Review of Psychology*, 31, 169–193.
- De Dreu, C. K. W. (2003). Time pressure and closing of the mind in negotiation. *Organizational Behavior and Human Decision Processes*, 91, 280–295.

- De Dreu, C. K. W. & Van Kleef, G. A. (2004). The influence of power on the information search, impression formation, and demands in negotiation. *Journal of Experimental Social Psychology*, 40, 303–319.
- DeSteno, D., Dasgupta, N., Bartlett, M. Y., & Caidric, A. (2004). Prejudice from thin air: the effect of emotion on automatic intergroup attitudes. *Psychological Science*, 15, 319–324.
- Dunn, J. R. & Schweitzer, M. E. (2005). Feeling and believing: The influence of emotion on trust. *Journal of Personality and Social Psychology*, 88, 736–748.
- Ekman, P. (1993). Facial expression and emotion. *American Psychologist*, 48, 384–392.
- Fischer, A. H. & Roseman, I. J. (2007). Beat them or ban them: The characteristics and social functions of anger and contempt. *Journal of Personality and Social Psychology*, 44, 362–369.
- Fisher, R. & Ury, W. (1981). *Getting to yes*. New York: Penguin Books.
- Fiske, S. T. & Dépret, E. (1996). Control, interdependence, and power: Understanding social cognition in its social context. In W. Stroebe & M. Hewstone (Eds.), *European review of social psychology* (Vol. 7, pp. 31–61). Chichester, England: Wiley.
- Forgas, J. P. (1995). Mood and judgment: The affect infusion model (AIM). *Psychological Bulletin*, 117, 39–66.
- Forgas, J. P. (1998). On feeling good and getting your way: Mood effects on negotiator cognition and behavior. *Journal of Personality and Social Psychology*, 74, 565–577.
- Forgas, J. P. (Ed.). (2001). *Handbook of affect and social cognition*. Mahwah, NJ: Erlbaum.
- Frank, R. H. (1988). *Passions within reason: The strategic role of the emotions*. New York: Norton.
- Fridlund, A. J. (1992). The behavioral ecology and sociality of human faces. In M. S. Clark (Ed.), *Review of personality and social psychology* (Vol. 13, pp. 90–121). Thousand Oaks, CA: Sage.
- Friedman, R., Anderson, C., Brett, J., Olekalns, M., Goates, N., & Lisco, C. C. (2004). The positive and negative effects of anger on dispute resolution: Evidence from electronically mediated disputes. *Journal of Applied Psychology*, 89, 369–376.
- Frijda, N. H. (1986). *The emotions*. Cambridge: Cambridge University Press.
- Frijda, N. H. & Mesquita, B. (1994). The social roles and functions of emotions. In S. Kitayama, & H. S. Markus (Eds.), *Emotion and culture: Empirical studies of mutual influence* (pp. 51–87). Washington, DC: American Psychological Association.
- Güth, W., Schmittberger, R., & Schwarze, B. (1982). An experimental analysis of ultimatum bargaining. *Journal of Economic Behavior and Organizations*, 3, 367–388.
- Hatfield, E., Cacioppo, J. T., & Rapson, R. L. (1994). *Emotional contagion*. New York: Cambridge University Press.
- Heider, F. (1958). *The psychology of interpersonal relations*. New York: Wiley.
- Isen, A. M. (1999). Positive affect. In T. Dalgleish & M. J. Power (Eds.), *Handbook of cognition and emotion* (pp. 521–539). New York: Wiley.
- Jones, E. E. & Davis, K. E. (1965). From acts to dispositions: The attribution process in social psychology. In L. Berkowitz (Ed.), *Advances in experimental social psychology* (Vol. 2, pp. 219–266). New York: Academic Press.
- Jones, E. E. & Harris, V. A. (1967). The attribution of attitudes. *Journal of Experimental Social Psychology*, 3, 1–24.
- Jones, E. E. & Nisbett, R. E. (1971). *The actor and the observer: Divergent perceptions of the causes of behavior*. New York: General Learning Press.
- Kelley, H. H. (1967). Attribution theory in social psychology. In D. Levine (Ed.), *Nebraska symposium on motivation* (Vol. 15, pp. 192–240). Lincoln: University of Nebraska Press.
- Keltner, D., Ellsworth, P. C., & Edwards, K. (1993). Beyond simple pessimism: Effects of sadness and anger on social perception. *Journal of Personality and Social Psychology*, 64, 740–752.
- Keltner, D. & Haidt, J. (1999). Social functions of emotions at four levels of analysis. *Cognition and Emotion*, 13, 505–521.
- Keltner, D. & Robinson, R. J. (1997). Defending the status quo: Power and bias in social conflict. *Personality and Social Psychology Bulletin*, 23, 1066–1077.
- Kennedy, D. B., Homant, R. J., & Homant, M. R. (2004). Perception of injustice as a predictor of support for workplace aggression. *Journal of Business and Psychology*, 18, 323–336.
- Klinnert, M., Campos, J., Sorce, J., Emde, R., & Svejda, M. (1983). Emotions as behavior regulators: Social referencing in infants. In R. Plutchik, & H. Kellerman (Eds.), *Emotion theory, research, and experience* (Vol. 2, pp. 57–68). New York: Academic Press.
- Knapp, A. & Clark, M. (1991). Some detrimental effects of negative mood on individuals' ability to solve resource dilemmas. *Personality and Social Psychology Bulletin*, 17, 678–688.
- Knutson, B. (1996). Facial expressions of emotion influence interpersonal trait inferences. *Journal of Nonverbal Behavior*, 20, 165–182.
- Kopelman, S., Rosette, A. S., & Thompson, L. (2006). The three faces of eve: An examination of the strategic display of positive, negative, and neutral emotions in negotiations. *Organizational Behavior and Human Decision Processes*, 99, 81–101.

- Kruglanski, A. W. (1989). *Lay epistemics and human knowledge: Cognitive and motivational bases*. New York: Plenum.
- Kruglanski, A. W. & Webster, D. M. (1996). Motivated closing of the mind. "Seizing" and "freezing." *Psychological Review*, 103, 263–283.
- Lerner, J. S. & Keltner, D. (2001). Fear, anger, and risk. *Journal of Personality and Social Psychology*, 81, 146–159.
- Lerner, J. S. & Tiedens, L. Z. (2006). Portrait of the angry decision maker: How appraisal tendencies shape anger's influence on cognition. *Journal of Behavioral Decision Making*, 19, 115–137.
- Loewenstein, G. F., Weber, E. U., Hsee, C. K., & Welch, N. (2001). Risk as feelings. *Psychological Bulletin*, 127, 267–286.
- Matsumoto, D. (1993). Ethnic differences in affect intensity, emotion judgments, display rule attitudes, and self-reported emotional expression in an American sample. *Motivation and Emotion*, 17, 107–123.
- Mayer, J. D., Salovey, P., Gombert-Kaufman, S., & Blainey, K. (1991). A broader conception of mood experience. *Journal of Personality and Social Psychology*, 60, 100–111.
- Messick, D. M. & Brewer, M. B. (1983). Solving social dilemmas: A review. In L. Wheeler & P. Shaver (Eds.), *Review of personality and social psychology* (Vol. 4, pp. 11–44). Beverley Hills, CA: Sage.
- Morris, M. W. & Keltner, D. (2000). How emotions work: An analysis of the social functions of emotional expression in negotiations. *Research in Organizational Behavior*, 22, 1–50.
- Oatley, K. & Jenkins, J. M. (1992). Human emotions: Function and dysfunction. *Annual Review of Psychology*, 43, 55–85.
- Parkinson, B. (1996). Emotions are social. *British Journal of Psychology*, 87, 663–683.
- Pillutla, M. M. & Murnighan, J. K. (1996). Unfairness, anger, and spite: Emotional rejections of ultimatum offers. *Organizational Behavior and Human Decision Processes*, 68, 208–224.
- Pinkley, R. L. (1995). Impact of knowledge regarding alternatives to settlement in dyadic negotiations: Whose knowledge counts?. *Journal of Applied Psychology*, 80, 403–417.
- Pruitt, D. G. (1981). *Negotiation behavior*. New York: Academic Press.
- Pruitt, D. G. & Carnevale, P. J. (1993). *Negotiation in social conflict*. Buckingham, England: Open University Press.
- Putnam, L. L. (1994). Challenging the assumptions of traditional approaches to negotiation. *Negotiation Journal*, 10, 337–346.
- Quigley, B. M. & Tedeschi, J. T. (1996). Mediating effects of blame attributions on feelings of anger. *Personality and Social Psychology Bulletin*, 22, 1280–1288.
- Rafaeli, A. & Sutton, R. I. (1987). Expression of emotion as part of the work role. *Academy of Management Review*, 12, 23–37.
- Roseman, I. J., Antoniou, A. A., & Jose, P. E. (1996). Appraisal determinants of emotions: Constructing a more accurate and comprehensive theory. *Cognition and Emotion*, 10, 241–277.
- Ross, L. (1977). The intuitive psychologist and his shortcomings: Distortions in the attribution process. In L. Berkowitz (Ed.), *Advances in experimental social psychology* (Vol. 10, pp. 173–220). New York: Academic Press.
- Schwarz, N. & Clore, G. L. (1983). Mood, misattribution, and judgments of well-being: Informative and directive functions of affective states. *Journal of Personality and Social Psychology*, 45, 513–523.
- Sinaceur, M. & Tiedens, L. Z. (2006). Get mad and get more than even: When and why anger expression is effective in negotiations. *Journal of Experimental Social Psychology*, 42, 314–322.
- Skarlicki, D. P. & Folger, R. (1997). Retaliation in the workplace: The roles of distributive, procedural, and interactional justice. *Journal of Applied Psychology*, 82, 434–443.
- Smith, C. A., Haynes, K. N., Lazarus, R. S., & Pope, L. K. (1993). In search of the "hot" cognitions: Attributions, appraisals, and their relation to emotion. *Journal of Personality and Social Psychology*, 65, 916–929.
- Smith, C. A. & Lazarus, R. S. (1993). Appraisal components, core relational themes, and the emotions. *Cognition and Emotion*, 7, 233–269.
- Steinel, W., Van Kleef, G. A., & Harinck, F. (2008). Are you talking to me?!? Separating the people from the problem when expressing emotions in negotiation. *Journal of Experimental Social Psychology*, 44, 362–369.
- Tiedens, L. Z. & Linton, S. (2001). Judgment under emotional certainty and uncertainty: The effects of specific emotions on information processing. *Journal of Personality and Social Psychology*, 81, 973–988.
- Van Kleef, G. A. (2009). How emotions regulate social life: The emotions as social information (EASI) model. *Current Directions in Psychological Science*, 18, 184–188.
- Van Kleef, G. A. & Côté, S. (2007). Expressing anger in conflict: When it helps and when it hurts. *Journal of Applied Psychology*, 92, 1557–1569.
- Van Kleef, G. A., De Dreu, C. K. W., & Manstead, A. S. R. (2004a). The interpersonal effects of anger and happiness in negotiations. *Journal of Personality and Social Psychology*, 86, 57–76.

- Van Kleef, G. A., De Dreu, C. K. W., & Manstead, A. S. R. (2004b). The interpersonal effects of emotions in negotiations: A motivated information processing approach. *Journal of Personality and Social Psychology*, 87, 510–528.
- Van Kleef, G. A., De Dreu, C. K. W., Pietroni, D., & Manstead, A. S. R. (2006). Power and emotion in negotiations: Power moderates the interpersonal effects of anger and happiness on concession making. *European Journal of Social Psychology*, 36, 557–581.
- Van Kleef, G. A., Steinel, W., Van Knippenberg, D., Hogg, M., & Svensson, A. (2007). Group member prototypicality and intergroup negotiation: How one's standing in the group affects negotiation behaviour. *British Journal of Social Psychology*, 46, 129–154.
- Webster, D. M. & Kruglanski, A. W. (1994). Individual differences in need for cognitive closure. *Journal of Personality and Social Psychology*, 67, 1049–1062.
- Weiner, B. (1995). *Judgments of responsibility: A foundation for a theory of social conduct*. New York: Guilford Press.

Chapter 32

Anger, Violence, and Political Science

Roger Petersen and Sarah Zukerman

Abstract Violence is one of the major topics of political science. Yet, due to its general failure to study the role of emotions, the discipline is not fully equipped to address key issues central to violence. For the study of political violence, anger holds special significance. Anger has a clear connection with motivations to commit or support violence. This chapter summarizes findings and methods in the study of anger in psychology and other fields and discusses how insights from these fields can be borrowed or modified to improve the study of violence in political science. The chapter's last section illustrates the usefulness of these hybrid concepts by applying them to an important concrete case – Colombia's current drive to demobilize combatants and reconcile its society in the face of a continuing civil war.

32.1 Introduction

Violence is one of the major topics of political science. Yet, due to its general failure to study the role of emotions, the discipline is not fully equipped to address key issues central to violence. examples of this shortcoming are readily available. Political scientists often describe a “hardening” of ethnic identity after violence among ethnic groups. They do not, however, really explain why and how this phenomenon happens or consider why or when identities might again “soften” (Van Evera, 2001; Kaufmann, 1996). Other political scientists discuss how political entrepreneurs instigate riots before elections in order to “heighten” ethnic salience as a tactic to change voting behavior (Brass, 1996). In another version of this tactic, insurgents use violent provocations, such as blowing up a religious site, in order to create tit-for-tat spirals of violence that escalate the conflict. In these actions, insurgents believe that they can generate a reaction in a target population, even if that reaction works against the long-term interests of that population. Furthermore, in dozens of works, political scientists address the subject of post-violence “reconciliation” among groups in conflict. As discussed in the material below, often the focus is on ending a desire for vengeance that follows in the wake of war.

Emotions are clearly relevant to all of the phenomena above. Moreover, the emotion of anger is central to the specific issues above. As discussed below, emotions can be partly defined by identifying cognitive antecedents and action tendencies. Anger is defined by appraisal that an individual or a

R. Petersen (✉)
Massachusetts Institute of Technology, Boston, MA, USA
e-mail: rpeters@mit.edu

group has committed an offensive action against one's self or group. The action tendency of anger is to punish that individual or group. The "hardening" and "heightening" of identities through violence and provocation, the retaliatory spirals of violence seen in many conflicts, the pursuit of "normal life" free of obsessions for vengeance all involve the central appraisals and action tendencies of anger.

This chapter develops a conceptualization of anger useful for the political scientist studying violence and its aftermath. The next section addresses the core nature of political violence and why political science has not been able to address many key elements of violent conflict. In practice, political scientists studying violence not only ignore anger but also generally avoid reference to emotions at all. The second section summarizes findings and methods in the study of anger in psychology and other fields and discusses how insights from these fields can be borrowed or modified to improve the study of violence in political science. The last section illustrates the usefulness of these hybrid concepts by applying them to an important concrete case – Colombia's current drive to demobilize combatants and reconcile its society in the face of a continuing civil war.

32.2 Political Violence and Political Science

In this chapter, we are most concerned with war and violence among groups within a state. The domain includes civil wars, riots, and internal ethnic conflicts. While emotions such as anger certainly affect criminal violence and may influence interstate wars, it is internal war that accounts for much of the world's violence today. Since the end of World War II, civil wars have probably killed five times as many as interstate wars (Fearon & Laitin, 2003). Clearly, variables such as economic inequality, imbalances in military force, and access to political institutions are linked to the outbreak, the length, and the termination of this violence. Yet, this set of standard structural variables fails to capture or address several salient and core qualities of political violence. A short list of these characteristics includes the following:

- (1) *Recognizable actors and actions.* Political violence generally involves recognizable actors (ethnic groups, political parties, insurgent groups, leaders) committing specific, purposeful, and blameworthy actions. For the participants, and especially the victims, of political violence, there is usually a clear idea that "group X committed this harmful action against group Y."
- (2) *Violence often takes place among groups with long-term relations.* "Group X and group Y" have likely interacted for years and will continue to interact in the post-conflict future.
- (3) *Elements of domination.* Violence introduces elements of domination and subordination into group relations.
- (4) *Intensity of experience.* The repressive actions, desecrations, killings, and bombings involved with political violence are likely to produce intense and possibly new experiences that disrupt normal life.
- (5) *Distortion of cognitive processes.* The intense experiences during and after violence often trigger mechanisms that distort information collection and belief formation.
- (6) *Elevated preferences.* Violence transforms and heightens specific preferences, in particular the desire for flight, retaliation, and vengeance.
- (7) *Changing intensity of preferences.* For example, during "hurting stalemates" or in the post-violence period, participants are likely to want to "move on with their lives" and their desire to continue violence or to punish the opponent may fade.

The first three elements emphasize the social nature of political violence. The next two relate to the intensity and disruption. The last two address preferences and their transformation and evolution during violence. These elements have various effects at different stages in the outbreak and course of violence and internal war.

As currently practiced, US political science is not well-equipped to address these realities of political conflict directly. The main reason for this problem is that current norms within the field privilege methods and explanations based on assumptions of narrowly rational actors operating according to a restricted set of preferences. This issue is obvious in the case of rational choice methods, perhaps less obvious in studies employing large-*n* regressions.

During the last two decades, rational choice models and large-*n* statistical models have been the dominant methods for studying violence. In examining major political science journals, rational choice (RC) models came into common use during the late 1980s. In rational choice, the individual is assigned one ordered preference structure (e.g., safety > revenge > self-esteem) and action can be predicted from the nature of constraints and incentives. If safety is not at issue, then the individual may pursue the secondary goal of revenge. RC relies on certain consistency requirements regarding preferences. Two are most fundamental. First, the agent must be able to rank order all choices (completeness). Second, the preferences must be transitive. Underlying these specific consistency requirements is a more general assumption that preferences are essentially stable. Furthermore, rationality assumes that agents are collecting an optimal amount of information and forming logical beliefs from that information.

Clearly, RC is not well-tailored to several of the qualities of political violence listed above. First and foremost is the issue of preferences (points 6 and 7). Economists regularly order preferences. However, it is one thing to assume that individuals prefer \$10 today to \$20 a year from now, or to draw a curve representing trade-offs in spending between military programs and social programs, and quite another to make assumptions about the relative values of such disparate desires as revenge, safety, and self-esteem and subordination that are so central to political violence. RC also has little to say about how the intensity of preferences might change over time or about when one goal might suddenly dominate all others to the point that trade-offs are no longer considered.¹

In RC, the stability of preferences is a simplifying assumption. Most practitioners of rational choice would probably agree that this simplification is not always useful for every type of human behavior. As many observers have noted, RC has produced its most useful insights in iterative situations or under stable institutional environments (as opposed to point 4 above). Both supporters and critics of RC agree with this view concerning the conditions appropriate for rational choice methodology. As one notable proponent of rational choice, George Tsebelis (1990) has summarized, “actions taken in noniterative situations by individual decision makers (such as in crisis situations) are not necessarily well-suited for rational choice predictions.” Furthermore, in a rather obvious point, RC is not interested in addressing the problems of cognitive distortions (point 5) as it simply assumes them away.

For many political scientists, rational choice methods failed to live up to expectations (Green & Shapiro) and in the past few years, there has been a turn toward addressing violence through large-*n* regressions that employ structural variables. David Laitin and James Fearon’s (2003) article on civil war and insurgency has been so influential in establishing discipline norms that it is used as an exemplar here. Their primary dependent variable is the onset of civil war. Based on a reading of other political science works, they develop a set of independent variables that includes level of gross

¹For an extended discussion of the role of emotions in economic theory, especially in light of their impact on preferences, see Loewenstein (2000).

domestic product, income inequality, nature of terrain, population size, ethnic and religious diversity, and extent of civil liberties. They find that the variables associated with grievances and identities are statistically insignificant, while those associated with level of GDP, terrain, and population size are statistically significant. Fearon and Laitin's interpretation of these findings is that civil wars are largely a matter of insurgent technology. Rebels fight for a variety of reasons but they do so only when they can rationally avoid capture by the state. They can challenge the state if they can hide in mountains or within large populations and if the state's capabilities are weak (proxied by GDP figures).

While these types of studies offer valuable accounts of the likelihood of war onset given structural conditions, they are limited in their ability to address the list of political violence characteristics above. The variables are necessarily coarse and static; they have difficulty engaging in the changing nature of social relationships in points 1–3. Fearon and Laitin's grievance variable is defined in terms of civil liberties and does not seriously try to capture the dynamics of dominance and group esteem that are fundamental to political violence.² Their rationalist interpretation clearly dismisses the discussion of cognitive processes and shifting preferences. In fact, the article suggests, by assuming the same motivations to be universally present, that scholars need not address motivations at all, let alone try to analyze specific emotions affecting violence.

Fearon and Laitin recognize the limits of large-*n* statistical studies and they also look to case studies and qualitative treatments of civil war as a check on their findings. However, Fearon and Laitin seem to transfer the rationalist assumptions underlying the regression findings to their treatment of this material. In a review of a set of largely anthropological and case study works on violence, they address the puzzle of why individuals appear to participate in communal violence when it does not appear rational to do so. They solve the puzzle by concluding that “‘ethnic violence’ can be a cover for other motivations such as looting, land grabs, and personal revenge; and the activities of thugs set loose by the politicians can ‘tie the hands’ of publics who are compelled to seek protection from the leaders who have endangered them” (2003, p. 874). Again, the explanation predictably seeks answers by positing a narrowly rational individual pursuing a constricted range of goods.

In a second highly influential cross-national quantitative study, Paul Collier (2003) and his collaborators conclude that civil war is overwhelmingly linked to economic variables. They find that political grievances and social divisions, inequality, and a host of other factors are not statistically significant; rather, a simple combination of accessible natural resources and a weak state produce civil war. These correlations are then interpreted in rational choice terms. The statistically significant variables are assumed to produce the constraints and incentives that affect the rational decisions of rebels in their pursuit of narrow interests, primarily economic goods. In this view, violence is a resource that is used to grab wealth. There are two versions of this “greed” theory. In one, the existence of natural resources provides a motive for conflict and war. In the other, the focus is on the lack of opportunities for legitimate economic activity in poorer, weaker states. Again, grievances and non-material motivations, let alone emotions, are not seen as productive avenues for explanation. In a passage on recruitment into rebel armies, Collier et al. address the question of non-economic motivation with the following speculation: “(T)he people who join rebel groups are overwhelmingly young uneducated males. For this group, objectively observed grievances might count for very little. Rather, they may be disproportionately drawn from those easily manipulated by propaganda and who find the power that comes from possession and use of a gun alluring. Social psychologists find

²Fearon and Laitin include variables concerning whether there is an official religion or not and whether linguistic and religious groups with over 5% of the population fail to receive official recognition or not. These variables cannot capture the dynamic processes involved in ethnic conflicts and they lump together disparate cases.

that around 3% of the population has psychopathic tendencies and actually enjoys violence against others (Pinker, 2002) and this is more than is needed to equip a rebel group with recruits" (Collier et al., 2003, p. 68). In Collier et al.'s approach, the actual everyday experiences of larger groups of people do not carry explanatory significance. Anger at violence, resentment of domination, historically and culturally based prejudices, and stigmas are not particularly relevant. Rather, violence is a matter of greedy elites operating according to structural constraints who lead a small set of naïve or psychopathic recruits.

In sum, recent political science approaches to violence have tended to employ blunt, static variables interpreted within a rationalist explanatory framework. There is a lack of a sense of process, especially in terms of how violence creates changing social relations.³ There is a general lack of specification of causal mechanism. Rational choice methods do identify a mechanism, but one not well-suited to address the nature of political violence. There is a lack of explanations based on non-rational mechanisms such as social norms, prejudice, cognitive distortions, and emotions.

Some movement away from this position can be observed. In an essay that includes a review of the political science literature on violence, Ronald Suny (2004) states: "I argue something that should be obvious, though not always for political scientists: emotions are key to human motivation. Indeed, we would not be human without them. They are a stimulus to action; they are fundamental to self-identification, to thinking about who "we" are and who the "other" is; they are involved in the social bonds that make groups, even whole societies, or nations, possible. And they are, therefore, powerful tools to explain why people do what they do politically." Several recent works have attempted to address the relationship between emotion and conflict (Kaufman, 2001; Petersen, 2002; Hyman, 2006; also Elster, 1999). It is still a short list. Suny is probably correct concerning the overall state of the study of emotions in political science and related fields when he concludes, "Far too often historians and other social scientists use explanations that emphasize emotions without specifying either that they are about emotions, which emotions are at play, and what the action tendency of those emotions is likely to be" (Suny, 2004).

Despite such urging, the inclusion of emotions into the study of political violence will accelerate only when the value-added of such inclusion becomes obvious. On the whole, political scientists seem to see emotions as too intangible and murky to be useful for a social scientific treatment of political violence. Many political scientists will ask if current models have good predictive power, then why should they try to incorporate emotion? There are two answers to this question. First, few would see the existing models as possessing strong predictive power.⁴ Second, the hypotheses derived from existing models have not been tested alongside hypotheses derived from models that include emotions. Competitors need to be developed through rigorous application of the scientific method. The scientific method involves accurate description of a phenomenon, specification of causal mechanisms, and formulation of testable hypotheses. An understanding of emotion, in this case the emotion of anger, can be highly useful, if not essential, to all three tasks in the study of political violence.

³A new wave of works concentrating on organization and the more dynamic processes of insurgency includes Kalyvas (2006), Weinstein (2007), and Wood (2003). Only Wood treats emotion (the emotion of pride) as a systematic factor.

⁴Collier and Hoeffler's (2004) model, for example, can only explain 20–30% of the variation in civil war onset.

32.3 Anger and Political Violence

If the description of political violence above is at all accurate, ignoring emotions impoverishes scientific analysis. The first step of the scientific method is observation and description of a phenomenon. Psychologists' theory and treatment of the emotion of anger describes and helps explain at least four of the seven characteristics of political violence listed above.

Emotion theorists commonly define and differentiate specific emotions by five characteristics: arousal, expression, feeling, cognitive antecedent, and action tendency. The cognitive antecedent of anger is that an individual or a group has committed a blameworthy action against one's self or group. The first point above is that political violence generally involves recognizable actors (ethnic groups, political parties, insurgent groups, leaders) committing specific, purposeful, and often reprehensible actions. For the participants, and especially the victims, of political violence, there is usually a clear idea that "group X committed this terrible action against group Y." As opposed to much criminal violence and some international conflicts, during internal political violence, the actors and their actions can be named. For example, Muslims in Bosnia can say "Serbs mass-murdered Muslims in Srebrenica" and residents in Colombia can say "the guerrilla group FARC committed a massacre in our town." In many of these cases, individuals might be able to name their neighbors as participants or supporters. It is these specific appraisals that underlie violent conflicts.⁵

The sixth point observes that a commonly observed effect of political violence is a desire for vengeance. In terms of emotion theory, the action tendency of anger is toward punishing the individual or the group that committed the harmful action. Under the influence of anger, individuals become "intuitive prosecutors" (Goldberg, Lerner, & Tetlock, 1999). That is, individuals tend to specify a perpetrator and then seek justice. Inextricably linked to this strong desire to punish is the need to blame an individual or a group. Anger lowers the threshold for attributing harmful intent. Angry people blame humans, not situations (Keltner, Ellsworth, & Edwards, 1993). Once angered, individuals "perceive new events and objects in ways that are consistent with the original cognitive-appraisal dimensions of the emotion" (Lerner & Keltner, 2000). This creates a positive feedback loop – anger increases blame which, in turn, increases anger (Lerner & Tiedens, 2006).

As anger feeds on itself, it may also persist through rumination. The mechanism is as follows: "When an emotion node is activated, past events and beliefs associated with that emotion are brought to mind, prolonging or increasing the emotion. Rumination or self-focus on the negative emotion should enhance this spreading activation and therefore exacerbate the emotion" (Rusting & Nolen-Hoeksema, 1998, 791). The cognition about the past anger episodes generates new episodes of state anger, amplifying the intensity and duration of the anger (Sukhodolsky, Golub, & Cromwell, 2001).

Addressing point five above (distortion of cognitive processes), the cognitive mechanisms associated with anger also enter into the course of the conflict. Critically, under the influence of anger, individuals reduce their estimates of risk and are more willing to engage in risky behavior (Lerner & Keltner, 2001; Gallagher & Clore, 1985; Mano, 1994; Lerner, Gonzalez, Small, & Fischhoff, 2003; Chapter 21 by D.M.T. Fessler, this book). Imbued with anger, combatants become overly optimistic in estimating the chances of successfully retaliating. These lowered estimates of risk therefore help create spirals of violence, with each side believing that they can successfully carry out their desired retaliation. Anger also heightens prejudice and locks combatants into stereotyping one another. Finally, in a point related to rumination, anger is capable of creating selective memory. Newhagen (1998) found that images producing anger were remembered better than those inducing fear, which

⁵It is worth noting that this understanding of the cognitive foundations of anger seems to be common across cultures. See Kassinvove, Sukhodolsky, and Tsytsarev (1997) on this point.

in turn were remembered better than those creating disgust. These selected negative images can become embedded into the developing narratives of a conflict.

Addressing the seventh point (changing intensity of preferences), psychologists have also studied the decline of anger. As Chapter 22 by M. Potegal, this volume points out, “any theory of anger must provide an account of temporal dynamics if it is to be considered complete.” Generally, social psychologists have found that anger rises quickly and then declines at a slower rate.

Psychologists have examined the ways in which the decay of anger might be accelerated through a study of “quenching mechanisms.” Quenching refers to a process that disrupts or dispels anger (Chapter 22 by M. Potegal, this book). It is commonly conceived of as a response to some extrinsic stimulus (a diversion), but can also be an intrapersonal mood regulation strategy: self-focused distraction (Rusting & Nolen-Hoeksema, 1998, 790). In essence, quenching refers to any thought, behavior, or external phenomenon that changes the emotional state, in this case, reduces the intensity of anger (Baumeister, 1991; Larsen, 1993; Morris & Rielly, 1987; Rusting & Larsen, 1993; Thayer, Newman, & McClain, 1994; Tice & Baumeister, 1993). If violent conflicts are sustained through anger and its effects, then the quenching of anger must be a major part of the termination of conflict and the reconciliation of its combatants.

It is important to emphasize that reactions to violence and victimization are extremely complex. At the time of the collapse of the Soviet Union, the senior author conducted dozens of interviews with survivors of some of the most brutal experiences of World War II and witnessed interplay of several emotions.⁶ These survivors experienced anger but also grief, fear, and pride (Petersen, 2005). There are also long-term issues of guilt and shame. Additionally, many victims clearly suffered from post-traumatic stress syndrome (PTSD). At the time of writing, the junior author is conducting a large-scale survey and in-depth interviews of demobilized paramilitaries in Colombia to evaluate their cognitions and reactions to violence.

While we accept that emotional responses to atrocity are likely multifaceted and complex, we focus on the emotion of anger for several reasons. First, survivors’ psychological traumas exist at the individual level of analysis and are not amenable to aggregation to the units of analysis with which political scientists are concerned: insurgent organizations, ethnic groups, states, etc. Second, violence to peace transition policies are built around notions of anger. While states seek to ensure healthy individual citizens (psychologically and physically), the dominant peace and reconciliation policies occur at a more aggregate level (that of a society) and assume an emotional logic that emphasizes anger, not other emotions. This is not to say that these other emotions (guilt, shame, grief) are not present or that they do not merit attention. However, there is no easy “quenching” of the emotional and psychological effects of violent conflict. Governments cannot easily address an issue like PTSD,⁷ but they often do seem compelled to address motivations for vengeance. For the study of political violence, anger holds special significance. Anger has a clear connection with motivations to commit or support violence. There is a clear imperative to reduce the intensity of this motivation. States may not be able to create the presence of factors required for deep reconciliation and reintegration, but they may be able to create an absence of intense anger.

As will be discussed in more detail in the empirical case below, post-conflict reconciliation is often based on a theory of how punishment, truth and apology, and reparations may act to reduce the corrosive effects of anger. These policies are based, implicitly, on theories of quenching anger. While

⁶These interviews were critical to Petersen (2001) and Petersen (2002).

⁷Although increasingly, demobilization and reintegration programs include a social-psychological dimension according to the logic that only psychologically stable individuals can reenter the work force and civil society successfully. The motivations for including social-psychological treatment are not to prevent violence or to encourage reconciliation.

polymakers move ahead with reconciliation policies, social scientists should follow by developing knowledge and hypotheses capable of testing the basic contours of these policies. Existing work on quenching of anger provides direction for this task.

32.3.1 Problems with the Psychology Approach to Anger

While the insights of psychologists have much to contribute to the study of political violence, many of the concepts cannot be directly applied without significant modification. While psychology does a good job in addressing the cognitive antecedents, action tendencies, strong and obsessive preferences, and preference change within political violence, most psychology treatments of anger cannot capture either the intensity of the experience of living through internal war or the rich, long-term social nature of political violence and its consequences.

Consider intensity first, the fourth point above. Although transient anger has been induced in any laboratory experiments with college students and some other accessible groups, these experiments cannot reproduce the feelings of atrocity survivors. As Jon Elster has summarized, “(B)ecause of the power of many emotions, there are limits to what we can learn from studies of human behavior under controlled conditions. Inducing strong emotions of love, shame, and hatred in the laboratory would not only be blatantly unethical but unfeasible. And there is no presumption that what we can learn from studying the milder forms of these emotions – liking, embarrassment, or disliking – will generalize to the more urgent or virulent forms” (Elster, 1999, p. 404). Psychology, as Elster further notes, identifies and specifies emotion-based mechanisms. Through the specification of these mechanisms, the laboratory provides the basis for creating hypotheses to be tested with data from violent conflicts even if the phenomena tested in the laboratory and the experiences in actual violent conflicts cannot be directly equated.

Second, addressing points two and three above, the emotions involved with political violence are acutely social. Unlike many laboratory experiments, real-life conflict is not a one-shot event. Consider the problem of post-violence reconciliation. David Cohen, working East Timor in its reconstruction period, tells the story of a widow who could not avoid seeing on a regular basis her husband’s killer wearing her husband’s jacket.⁸ While most experiences are not so dramatic, in many instances of political violence, members of combating ethnic groups must intermingle in the post-violence period. Members of victim groups may have to buy something from a member of a perpetrator group. Even in situations where warring factions have been separated, groups may be looking across a river or a barrier and being reminded of previous atrocities. After being told that a Sunni family had moved into their family’s home, a displaced Shiite responds in an interview, “I try to imagine my room and what they do in it” (NYT, Tavernise). Often, cultural symbols take on new meanings and power. In the divided city of Mitrovica in Kosovo, for instance, Serbs have built a church high on a hill on their side of the river; the Albanians on the other side of the river cannot help but see it, and see it in light of the Serbs’ previous political dominance and acts of ethnic cleansing. A similar situation exists in the divided city of Mostar, Bosnia, with Croats and Muslims erecting religious structures at the boundary lines.

The experience of anger in these situations is something between rumination, discussed above, and constant new, but similar, evocations of anger. Memory of past atrocities mingles with daily experience. It is not clear that social science presently possesses the language to communicate this

⁸Story told at the Conference “Peace and Accountability in Transitions from Armed Conflict,” 15–16 June 2007, Bogotá, Colombia.

experience, but it is close to anger in its overall contours. The cognitive antecedent and action tendencies are basically the same. Similar to anger, the intensity of the experience seems to decay over time.

The one area in which anger needs radical redefinition from its common use in psychology is in terms of temporal dynamics, especially duration. In laboratory settings, psychologists measure the duration of emotions in terms of seconds and minutes. Chapter 22 by M. Potegal, this volume estimates that “a half hour is the modal duration of everyday anger.” Some episodes of anger last longer. For example, “A longitudinal study of consumers who had suffered service failures for which they consider getting even, found that anger had a half-life of more than a couple weeks” (Gregoire & Tripp, 2006, cited in Tripp and Bies). If anger lasts this long with respect to a service failure, then the death of a family member during a violent conflict should trigger a much slower decay of anger. Given the intensity of the experiences of political violence and the way these experiences connect to existing social groups and hierarchies, anger created through violent political conflicts must be treated in months and years instead of minutes and days.⁹

Finally, as noted above, psychologists have often found that anger quickly rises and then decays at a much slower rate. It is not clear that this particular curve will also be found in events associated with political violence. At the current level of understanding, we can only speculate on what such curves would look like. As shown below, assumptions about the temporal dynamics of anger are actually at the heart of crucial policy questions.

32.3.2 Developing an Analytical Framework Based on an Understanding of Emotion and Anger

We have been working toward a hybrid concept of anger that can generate testable hypotheses relating to political violence. This conceptual framework should be able to speak to the concerns and methods of political scientists. Ideally, the framework should help address major policies working to reduce violence or facilitate post-violence reconciliation. This section delineates which elements of anger, and by extension, emotion in general, should be emphasized within this framework.

An emotion is a complex of phenomena composed of cognitive antecedents, action tendency, specific cognitive mechanisms, as well as the immediate physical manifestations related to arousal, expression, and feeling. First of all, cognitive antecedents and action tendencies will likely be key accessible characteristics to define and differentiate emotions. (The physical manifestations of emotions are likely to be more relevant to voting and the effects of political advertising.). Second, some of the cognitive mechanisms associated with a particular emotion will be much more relevant to political violence than others. For example, one of the core models of conflict in political science is “the spiral model” which explains how retaliatory cycles of escalation and violence are initiated and maintained. The question arises as to why actors believe they can successfully win in these escalations. Clearly, mechanisms relating to beliefs about risk are important for this issue. Another puzzle for political scientists is the “hardening of identities” that occurs during conflict. On this issue,

⁹This is not to suggest that we merely rename the time axis with months and years instead of minutes and days; rather, as will be discussed below, we must reconceive the anger curves following atrocities. One way to imagine the graph would be as convex functions with survivors’ anger depreciating daily (in accordance with the dominant psychological findings), but then also being re-elicited daily by a combination of memories and rumination which returns the individuals’ anger to elevated levels. These peak daily levels, however, would diminish over time as the original anger-provoking event fades and therefore produces weaker cognitions and ruminations that, in turn, elicit weaker emotional responses.

knowledge of mechanisms relating to blaming and stereotyping is most crucial. It is worthwhile to break down an emotion into its sub-phenomena and then build hypotheses upon the most relevant mechanisms and aspects of that emotion. Third, how the emotion decays will be important.

In keeping with our “value-added” approach to including emotions, Fig. 32.1 illustrates a rational action model. Starting on the right side of Fig. 32.1, individuals are seen as holding a short list of stable and ordered preferences or desires. Given these desires, individuals then collect information about how best to attain their goals. They form beliefs about the most effective means and strategies to gain what they want. An action then Results as a combination of desires and beliefs. This cycle is of course oversimplified but does capture the basic elements of the rational choice approach summarized earlier.

Fig. 32.1 Action cycle with no reference to emotion

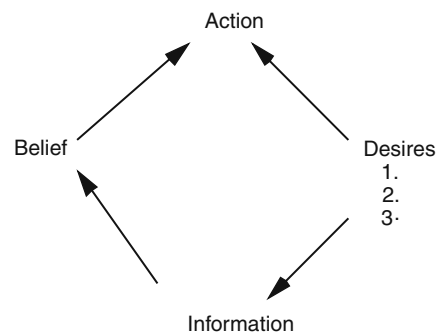


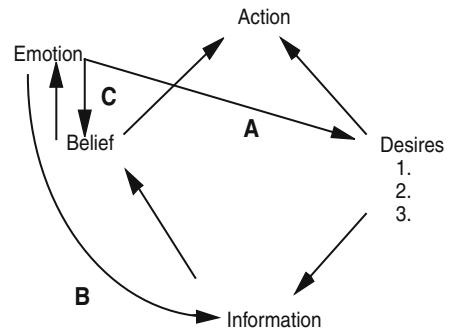
Figure 32.2 incorporates Fig. 32.1 but in this cycle, belief also leads to emotion. Following many socially oriented theorists, emotion can be conceptualized as “thought that becomes embodied because of the intensity with which it is laced with personal self-relevancy.” (Franks & Gecas, 1992) As Ortony, Clore, and Collins (1988) write: “Our claims about the structure of individual emotions are always along the lines that *if* an individual conceptualizes a situation in a certain kind of way, *then* the potential for a particular type of emotion exists.”¹⁰ In Fig. 32.2, belief also leads to emotion. For example, a belief about threats can lead to fear. A belief about status inconsistency can lead to resentment. A belief about the lack of worth of an object or an individual can lead to contempt. As discussed here, belief that an individual or a group has committed a blameworthy action against one’s self or group leads to anger.

Three general effects of emotion may follow, marked as A, B, and C effects in Fig. 32.2. Many of these effects have been mentioned above. First, and most fundamentally, emotions are mechanisms that heighten the saliency of a particular concern (A effect). This effect is closely related to action tendency. The emotion acts as a “switch” among a set of basic desires. Individuals may value safety, money, vengeance, and other goals, but emotion compels the individual to act on one of these desires above all others.¹¹ This effect may shape preferences lexicographically or it may operate by shaping the trade-offs among specific preferences (Elster, 1998). Emotion creates an urgency to act on a

¹⁰One of the biggest debates in emotion theory is the relationship and ordering of beliefs and emotion. Clearly, the relationship works both ways as is clear in Fig. 32.2 and its B and C effects. This issue is covered by several other chapters in this volume and we cannot do justice to it here. In other works, I have treated emotion as preceding beliefs. See the treatment of the emotion of rage in Petersen (2002).

¹¹The most influential work on the instrumental value of emotion in selecting among desires is probably Antonio Damasio’s (1994).

Fig. 32.2 Action cycle illustrating three possible effects of emotion



particular desire; the value of future pay-offs on other preferences is discounted; particular issues become obsessions. The emotion of anger heightens the desire for punishment and vengeance.

Second, once in place, emotions can produce a feedback effect on information collection (B effect). Emotions lead to seeking of emotion-congruent information. For example, individuals under the influence of fear may come to obsess about the chances of catastrophe. They may concentrate only on information stressing danger and ignore information about the lack of threat.

Third, emotions can directly influence belief formation (C effect) (Frijda, Manstead, & Bem, 2000). Emotions can be seen as “internal evidence” and beliefs will be changed to conform to this evidence. Even with accurate and undistorted information, emotion can affect beliefs. The same individual with the same information may develop one belief under the sway of one emotion and a different belief under the influence of a different emotion.¹² Furthermore, the style of belief formation may change under the grip of emotion. As political scientist William Riker has pointed out, rational individuals may operate according to several different sorts of strategies (“sincere,” “avoid the worst,” “average value,” “sophisticated”) (Riker, 1986, p. 26). For example, it is likely that emotions such as fear can influence a switch in method of belief formation, perhaps to an “avoid the worst” strategy.

With this general framework in mind, the specific emotion of anger can be summarized. Anger forms from the belief that an individual or a group has committed an offensive action against one’s self or group. Its A effects heighten desire for punishment and vengeance against a specific actor. Under the influence of anger, individuals become “intuitive prosecutors” specifying perpetrators and seeking vengeance (Goldberg et al., 1999). Anger’s B effects distort information in predictable ways. The angry person lowers the threshold for attributing harmful intent; the angry individual blames humans, not the situation (Keltner et al., 1993).¹³ Anger tends to produce stereotyping (Bodenhausen, Sheperd, & Kramer, 1994). Anger’s C effects shape the way individuals form beliefs. Under the influence of anger, individuals lower risk estimates and are more willing to engage in risky behavior (Lerner & Keltner, 2001; Gallagher & Clore, 1985; Mano, 1994; Lerner et al., 2003). In sum, regarding the key sub-phenomena of anger in relation to political violence, anger heightens desire for punishment against a specific actor, creates a downgrading of risk, increases prejudice and blame, as well as selective memory (Newhagen, 1998).¹⁴

¹²Also, the complete lack of emotion certainly affects information and belief formation. See the work of Damasio and others with brain-damaged patients who have lost their capacity for emotion.

¹³Keltner, Ellsworth, and Edwards studied angry subjects compared to sad subjects, asking both groups to interpret agency in an ambiguous event. Sad subjects assigned blame to the situation, angry ones to the actors.

¹⁴Newhagen found that images producing anger were remembered better than those inducing fear, which in turn were remembered better than those creating disgust.

Before discussing hypothesis formation, the temporal dynamics of emotion, especially anger, must be discussed. One of the essential questions of political violence is post-violence reconciliation. Closely related to this issue is whether negative emotions closely connected to the violence will recede in time and whether there are policies which will accelerate decay. How might emotions connected to political violence, such as anger, fade over time? It is possible to draw curves representing possible half-life functions of anger. Currently, social scientists possess little research that allows us to draw such functions for issues involved with political violence; however, some conjectures are possible. Figures 32.3, 32.4 and 32.5 represent different emotion curves. The vertical axis represents the intensity of the emotion. The horizontal axis represents time. As has been discussed, the time measure must be drawn in months and years. In Fig. 32.3, the intensity of the emotion declines in a linear fashion over time.¹⁵ Fig. 32.4 illustrates a situation of exponential decay in which the emotion is initially high but then decays rapidly. Figure 32.5 represents an inverse exponential relationship in which anger remains high for a long period and then declines at increasing rates.

For those studying political violence, one crucial question, as seen in the empirical case below, is whether policies can be created to change the shape of these temporal dynamics. This issue

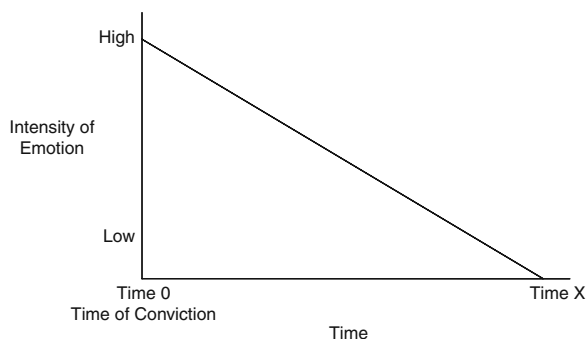


Fig. 32.3 Linear decline of emotion

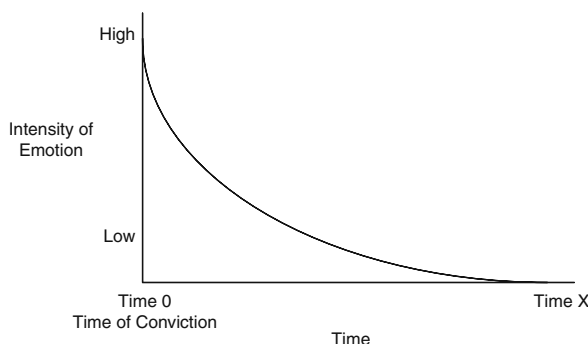
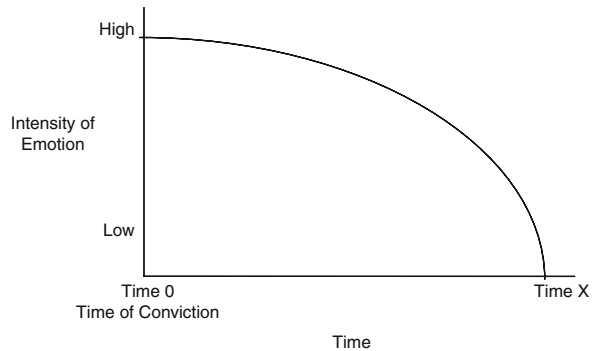


Fig. 32.4 Exponential decay of emotion

¹⁵As mentioned in an earlier footnote, another way to draw this curve would be as a series of convex functions whose maximums decrease linearly over time; that is, every day (or at an alternative frequency), some memory, cognition, or event re-evokes the original anger-causing event (be it the imagining of what a Shiite is doing in your home or seeing your husband's killer wearing your husband's coat). The result is a spike in the raw emotion of anger, which then declines in the order of minutes (or hours) until the next re-evoking event or thought. However, the spikes in anger likely also diminish over time. Three or ten years following the atrocity, the cognition is unlikely to elicit the same emotional response as 3 or 10 days after.

Fig. 32.5 Inverse exponential decay of emotion



brings us back to the study of “quenching mechanisms” mentioned above. In fact, many governments around the world have been developing policies connected, at least implicitly, to the erosion of anger. South Africa’s Truth and Reconciliation Commission is the most well known, but similar programs can be found in many Latin American and African states. These policies often see a combination of punishment, truth-telling, and apology as essential to reconciliation. In emotion-theory terms, these actions serve as quenching mechanisms. Knowledge of anger theory and the intertwined study of quenching help make explicit the causal logic often remaining implicit in these policies.

For victims, punishment facilitates quenching in several ways. First, violence creates an inequality between victim and perpetrator (addressing political violence point number three). The state’s vengeance acts to equalize this unbalanced power relationship. The victim is no longer the inferior one, the one to whom things can be done, the helpless and the object of someone’s arbitrary action. Vengeance also creates a sense of one’s power and control. Closely linked to power equalization is the restoration of threatened or damaged social prestige or self-esteem. Atrocities often attack a victim’s very sense of personal value and identity and vengeance enables the victim to reassert him/herself. One’s identity, in some cases, is so intimately linked to the esteem of a group that offenses against the group will also evoke strong desires for revenge and will give revenge much of its emotional force (see Frijda, 1994; Tajfel & Turner, 1979). Revenge also takes away the prospect of the perpetrator leading a happy life while one suffers. So the victim, through vengeance, accrues the benefit of taking away the offender’s gains. The victim gets “even in suffering.” Last, for family/friends of victims, revenge is a means to make their close-ones’ deaths meaningful, to keep faith with the dead, and to honor their memory. Revenge further serves to assign responsibility and thereby “relieve the moral ambiguity and guilt survivors often feel.” Finally, victims also use revenge to externalize their grief and bring closure (Hamber & Wilson, 2002).

Truth may quench anger by reducing the extent of error in the appraisal of perpetrators’ intentions. Victims seek to understand harm doers’ intentions so as to calculate the just response. However, they “make attribution errors about the harm doer’s motives. . . All such errors cumulatively add up to sloppy detective work and excessively blaming the harm doers, believing that the harm doer’s intentions were more intentional and personal than they actually were” (Tripp & Bies, 7). Truth thus cleans up some of the detective work and provides more information as to “why” the event occurred and the offender’s level of responsibility for offense (Kelley 1972, cited in Tripp and Bies). Since revenge requires blaming someone, truth, by changing the causal analysis of what happened, can reduce desires for revenge.

Related to truth is the notion of apology. When offenders apologize, anger, the desire for revenge, and levels of punishment are hypothesized to diminish (Weiner, Graham, Peter, & Zmuidinas, 1991; Bennett & Earwaker, 1994; Darby & Schlenker, 1989; Gonzalez, Haugen, & Manning, 1994; Holtgraves, 1989; Scher & Darley, 1997).¹⁶ The causal processes are fourfold. First, by exhibiting the emotions of sorrow, sadness, regret, shame, or guilt, the offender demonstrates to the victim his/her humanity which enables the victim to overcome stereotypes brought on by anger. Second, the apology produces a separation between the offender and his negative action; the offense is shamed, but the perpetrator is not. In this way, the perpetrator's inherent self-worth is redeemed and s/he becomes potentially worthy of restored relations and reconciliation with the victim (Petrucchi, 2002). In experimental work, evidence points to a relationship between offenders' apologies and victims' improved impression of their offenders and subdued feelings of aggression (Ohbuchi, Kameda, & Agarie, 1989). Third, the truth-telling and offered apology helps shift blame from the victim to the perpetrator. Victims often feel responsible for the offense or the atrocity. They suffer guilt, as they believe they could and should have prevented the offense. According to Petrucci (2002), learning that the atrocity's occurrence was not in their control through the apology "place[s] the responsibility of the act [back] on the shoulders of the offender."¹⁷ Last, apologies often comprise some type of offer of compensation, repair, or restitution, which serves as a power and status equalizer between the victim and the perpetrator. At the same time, throughout the apology offer and apology acceptance process, the victim remains in control (Abel, 1998). The literature indicates that the apology's timing (sooner is better), delivery (private and face-to-face preferable), believability, and the severity of the offense impact the apology's effectiveness at quenching anger (see Tavuchis, 1991; Petrucci, 2002).

The question is whether the framework above and its specification of mechanisms can help formulate hypotheses relevant to actual cases of political violence. We thus turn to an actual case, Colombia's civil war and its efforts to demobilize, to illustrate how testable hypotheses might be formulated from this theoretical framework.

32.4 Application to Colombia¹⁸

Over the past four decades, the Colombian conflict has touched every region of the country. In the past 20 years alone, violence has taken the lives of at least 70,000 people, internally displaced 3.5 million, and tortured, "disappeared," and kidnapped tens of thousands (CINEP, 2004a, b, 2005; Amnesty International, 2005). This violence has been committed by a variety of groups, not only by guerrillas, urban militias, criminals, and narco-traffickers but also by paramilitary groups with at least tacit linkages to the military.

¹⁶One might wonder if offenders wish to apologize and if victims wish to accept the apologies and forgive. Evidence shows that indeed offenders are interested in apologizing (Schlenker & Darby, 1981; Fercello & Umbreit, 1998) and that victims only very rarely reject apologies (Bennett & Dewberry, 1994; Bennett & Earwaker, 1994).

¹⁷Rusting and Nolen-Hoeksema indicate some preliminary findings that "it may be much more difficult to distract from angry thoughts and feelings in the heat of an unresolved situation than later after the situation has been somewhat resolved or the person has found a way to 'live with' the past event" (1998, 801). Truth and apology can potentially resolve situations and therefore bring closure.

¹⁸This section is from Roger Petersen and Sarah Zukerman's *Revenge or Reconciliation: Theory and Method of Emotions in the Context of Colombia's Peace Process*, 2009 in the Peace Research Institute of Oslo's Forum for International Criminal Justice and Conflict publication series. Spanish version to be published in Colombia by the Vice Presidency and the Universidad del Rosario Press.

Repeated violence across a long period of time develops its own local life. This was especially true of an earlier period of Colombian history, referred to as “La Violencia” (1948–1958), a period in which Conservatives and Liberals killed each other in deadly, and often local, spirals. As Robin Kirk (2003, p. 25) summarizes:

These were not crimes between strangers, but acts of astonishing violence between people who had known each other their whole lives. Called “La Violencia,” the struggle that rapidly consumed Colombia, was personal. Grand political fortunes were at stake, but so too were simmering land disputes, municipal rivalries, indiscretions, ambitions, and the affairs of the heart and gonads. Most of the killers were town men or of peasant stock, immersed in a world little different than that of their parents, grandparents, or even great-grandparents. So were the victims. The people who killed often knew their victims well, had known them since childhood, and had even been playmates, friends, family or neighbors.

Once blood had been shed, it was answered with more blood, in a spiral that devoured whole families. Vengeance is a theme that runs deep and true through Colombian history, the “scorpion in the breast,” to quote Colombian novelist Jose Eustacio Rivera, that “stabs at any instant with its stinger.” People killed to pay back other killings, to even the score left by Gaitan’s death, the War of a Thousand Days a half century earlier, the loss of land, of pride, of control. Often, killers left notes claiming responsibility for atrocities, ensuring that survivors were clear on their authorship.¹⁹

These local dynamics reappear in recent examples and data. León (2005) tells the story of Barrancabermeja, a typical Colombian town that has suffered waves of killing and counter-killing. First, it became an “incubator” of the ELN guerrillas in the 1980s that infiltrated the lower class neighborhoods, local politics, and the unions. In response to this “dangerous” symbiotic relationship between the ELN and the local population came brutal police repression at levels incommensurate with the scale of the strikes and protests. Indiscriminate repression in turn drove angry civilians into the arms of the ELN and the FARC who consolidated control over the region. Then, in 2001, the paramilitaries stormed Barranca, killing hundreds as they seized control over the territory and punished, in waves of reprisals, all civilians suspected of sympathizing with the guerrillas.²⁰

In each round of offensives, there are fatalities and displacement that generate a new population of victims. Some of these are impelled to take their desire for revenge and justice into their own hands. Figures can also be broken down by localities or groups. In Medellín, 25% of those joining the paramilitary Bloque Cacique Nutibara did so for reasons of personal revenge related to the death of a loved one. Another 25% joined due to external threats. Only 23% joined for economic reasons (Villegas, 2005).

Cognizant of the need to break these vicious cycles of killing, the Colombian government has embarked on a process of demobilization and reconciliation founded on law number the Justice and Peace Law. The law calls for a three-pronged process of truth, reparations, and punishment. On truth, individuals must make a full and honest confession of their actions in order to receive the full benefits and leniency of the law. On reparations, a newly created court establishes both monetary and symbolic compensation.

While truth and reparations are highly significant innovations in this reconciliation process, punishment is central. First, it represents a complete reversal of past policy. In the past, combatant leaders were enticed into laying down their arms with unconditional offers of amnesty which became a cycle

¹⁹In this work, Kirk argues passionately against seeing Colombia’s violence as resulting from a specific national “culture of violence.” Our position is that anger and the desire for revenge are a natural part of human nature and are found across a wide set of cases and time. For instance, the Law of Talion and innumerable instances of revenge in literature and religion (Medea, Oresteia, Hamlet, Tess, Cain’s killing of Abel, God’s expulsion of Adam and Eve, “an eye for an eye”) and in politics (in Corsica, the Balkans, Sudan, feudal Japan, and the southern United States) attest to the power and universality of the desire for vengeance. See, for example, Gould (2000).

²⁰Four hundred and three homicides were reported in Barranca in 2001.

of conflict followed by amnesty, then reinsertion, then conflict again (see Chernick, 1999).²¹ Today, Colombian political leaders emphasize that there can be no impunity, rather reconciliation demands punishment. Colombian leaders have tied their hands on this issue by allying with international human rights organizations.

Why have Colombians come around to the position that punishment is necessary to break cycles of violence? While some of the answer has to do with creating the conditions for future deterrence, the impulse toward punishment seems more based on an intuitive understanding that punishment, the diminishment of anger, and justice are all inextricably linked.

32.5 Generating Hypotheses from a Conceptual Framework of Anger

The state's policy of punishment can be seen in terms of a process connected to Fig. 32.2. In a first cycle, atrocities and violence create the cognition of anger: an individual or a group has committed an offensive action against one's self or group. The resulting emotion of anger greatly elevates a desire for retaliation and shapes information collection and belief formation. In effect, the state's actions create another turn of the cycle and add new information and beliefs. After a conviction of the perpetrators, the victim now holds the belief that an individual or a group has committed a negative action against one's self or group and that the state has put the perpetrator in prison.

This new set of appraisals may produce a lower intensity of the emotion of anger and its A, B, and C effects. In turn, the victim engages less in blaming and stereotyping. Victims are likely to assess the risks of retaliation more accurately and more soberly consider the costs of taking matters into their own hands. Victims may become less obsessed with the past and more oriented toward the future. In short, both punishment of the offenders and the passage of time may reduce anger.

Few in Colombia have considered how the policy might work over time. The nature of the erosion of the emotion is uncertain but Figs. 32.6–32.7 suggest some possibilities. For instance, punishment may change the values on the vertical axis (Fig. 32.6). Assume time 0 represents the perpetrator's date of conviction. Knowing that he will certainly be punished, the victim's anger drops immediately. Then the intensity of the emotion may decline according to the same function. The overall result, in this conception, is both a lower overall level of anger and a shorter life span of the emotion.

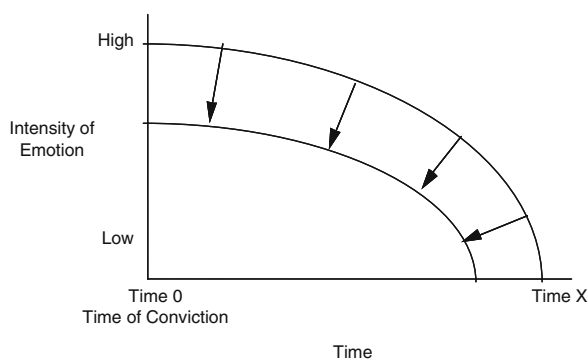
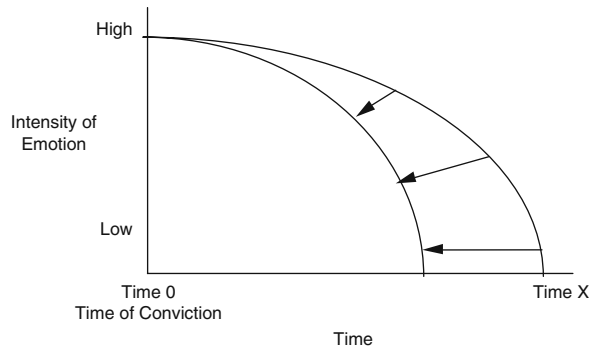


Fig. 32.6 A possible effect of punishment on the intensity of anger

²¹Law 35, operative 1981–1986, was the law of “olvido y perdón en pro de la paz” (law of forgetting and forgiveness in favor of peace).

Fig. 32.7 Possible effects of punishment on time effects of anger



A second possible effect of punishment might be compressing the horizontal axis, or the amount of time needed for the decay of anger. Time erodes anger. But how much time? If the perpetrator is punished, then anger may fade in 5 years rather than 10. Then the curve might look like Fig. 32.7. While the original intensity might remain high even at the time of conviction, the rate of decay accelerates.

Third, time of decay might remain the same, but punishment might change the shape of the curve. With convictions, anger's half-life might switch from Fig. 32.5 to Fig. 32.4 or Fig. 32.3.

The point to be made here is that the specification of anger mechanisms and the conjectures regarding its temporal dynamics help identify a set of variables and suggest possible causal relationships among those variables. The dependent variable here is the intensity of anger. The primary independent variables are the level of punishment, the passage of time, and the level of atrocity. These hypotheses might include the following:

Hypothesis 1: Higher levels of atrocity and violence will produce higher levels of anger.

Hypothesis 2 (general form): A significant level of punishment combined with the passage of time will reduce the level of anger.

Sub-hypotheses:

- (2A) From the time of conviction, the decline of anger will be linear (Fig. 32.3).
- (2B) From the time of conviction, the decline of anger will be exponential (Fig. 32.4).
- (2C) From the time of conviction, the decline of anger will be reverse exponential (Fig. 32.5).
- (2D) At the time of conviction, the level of anger will drop precipitously and then decline according to one of the functions in 1A–1C (Fig. 32.6).
- (2E) A significant level of punishment will not produce an immediate drop in the level of anger but will reduce the total life of anger (Fig. 32.7).

Further hypotheses can be linked to the other elements of the reconciliation law – truth and reparations. These are listed below along with a brief explanation of their causal logic:

Hypothesis 3: If reparations are added to punishment, anger will erode at an enhanced or accelerated rate.

Causal logic: Reparations are another form of punishment, in monetary terms rather than in prison time. There is a direct element of vengeance also, as resources are taken directly away from the perpetrator and given to the victim. This process bolsters the sense of equalization

of victim and perpetrator. Material reparations can “open space for bereavement, addressing trauma, and ritualizing symbolic closure. . . can [further] concretize a traumatic event and re-attribute responsibility” (Hamber & Wilson, 2002).

Hypothesis 4: If perpetrators confess to their crimes, if there is the addition of “truth” to punishment, anger will erode at an enhanced or accelerated rate.

Causal logic: In the transitional justice literature, it is hypothesized that truth-telling enhances the mitigating effect of “justice” on anger. It does so by constructing a common story of the past, honoring victims, breaking impunity, facilitating punishment of the guilty, and preventing the atrocities’ repetition. It is important to note that, for those who wish to know the truth, knowledge of the offender’s identity and motivations impacts levels of anger not via the ability to know whom to punish but through a different mechanism: by altering the information available to the victim. Learning the perpetrators’ motives and circumstances can undo the distorting effects of anger on information and beliefs; that is, by individualizing the perpetrator and showing his/her humanity, truth confessions can enable the victim to overcome stereotypes brought on by anger. If anger impels the victim to increase his/her prejudices and assignment of blame, remember selectively, and desire revenge, then truth, by providing new information, can alter the victim’s cognition that the perpetrator committed a harmful action against him/her. It thereby enables the victim to understand and forgive (see Gibson, 2004; Mockus, 2007):²² the act of removing the attribution of harmful intent from the offenders. In these ways, truth reduces anger.

The question remains as to if these hypotheses can be realistically tested in a case like Colombia. To our knowledge, no one has attempted to do so. A minimum requirement is that each of the variables – level of anger, level of atrocity, level of punishment, passage of time, reparations, and truth-telling – be operationalized in a realistic and reliable way. It is beyond the scope of this chapter to address this second stage of the scientific method – theory testing – but we seek to evaluate them elsewhere (Petersen & Zukerman, forthcoming).²³

32.6 Conclusions

Our goals in this chapter have been threefold. First, we aimed to show the relevance of the emotion of anger for the study of political violence. Perhaps surprisingly, political scientists seldom make reference to anger. Second, we have shown how findings from psychology and related fields can be used to enhance the description of political violence, to specify relevant mechanisms, and to develop a framework that generates hypotheses. Far from being a flight from science, reference and knowledge of emotions can enhance the scientific approach to political violence. Third, we have shown

²²More generally, some political figures have posited that only with truth can there be true forgiveness which in turn may reduce anger. This relationship between truth and forgiveness has been a central issue in the reconciliation process in South Africa. Gibson’s work shows that the acceptance of a common narrative of apartheid created positive effects concerning the legitimacy of the post-apartheid government and the acceptance of the rule of law. The role of forgiveness in reconciliation has been emphasized by the former Mayor of Bogotá, Antanas Mockus. In a talk titled “Why Forgiveness?” (Seminario Internacional Paz y Responsabilidad en la Resolución de Conflictos, Bogotá June 16, 2007), Mockus concentrated on the relationships between the emotions of guilt and shame but implied that pardon also reduces anger.

²³The authors have attempted to tackle this difficult issue in another forum. See Roger Petersen and Sarah Zukerman (2009).

how an understanding of anger and an analytical framework developed from that understanding can help develop methods to evaluate an important, ongoing violent conflict.

The incorporation of emotions such as anger into the study of political violence is challenging, but the pay-offs can be substantial.

References

- Abel, R. L. (1998). *Speaking respect, respecting speech*. Chicago: University of Chicago Press.
- Amnesty International. (2005) "Colombia: The Paramilitaries in Medellín: Demobilization or Legalization?"
- Baumeister, R. F. (1991). *Escaping the self*. New York: Basic Books.
- Bennett, M. & Dewberry, C. (1994). 'I've said I'm sorry, haven't I'? A study of the identity implications and constraints that apologies create for their recipients. *Current Psychology*, 13, 10–20.
- Bennett, M. & Earwaker, D. (1994). Victims' responses to apologies: The effects of offender responsibility of offense severity. *The Journal of Social Psychology*, 134, 457–464.
- Bodenhausen, G., Sheperd, L., & Kramer, G. (1994). Negative affect and social judgment – the differential impact of anger and sadness. *European Journal of Social Psychology*, 24, 45–62.
- Brass, P. (1996). *Riots and Pogroms*. New York: New York University Press.
- Chernick, M. (1999). Negotiating peace and multiple forms of violence: The protracted search for a settlement to the armed conflicts in Colombia. In C. J. Arnson (Ed.), *Comparative peace processes in Latin America*. Stanford: Stanford University Press.
- CINEP. (2004a). *Deuda con la Humanidad: Paramilitares del Estado en Colombia 1988–2003*. Bogotá: CINEP.
- CINEP. (2004b) *Revista Noche y Niebla*, No. 30.
- CINEP. (2005) *Revista Noche y Niebla*, No. 31.
- Collier, P., Elliott, V. L., Hegre, H., Hoeffler, A., Reynal-Querol, M., & Sambanis, N. (2003). *Breaking the conflict trap: Civil war and development policy*. Washington, DC: Copublication of the World Bank and Oxford University Press.
- Collier, P. & Hoeffler, A. (2004) "Greed and Grievance in Civil War," *Oxford Economic Papers*, Oxford University Press, 56(4), 563–595.
- Damasio, A. (1994). *Descartes' error: Emotion, reason, and the human brain*. New York: Quill.
- Darby, B. W. & Schlenker, B. R. (1989). Children's reactions to transgressions: Effects of the actor's apology, reputation and remorse. *British Journal of Social Psychology*, 28, 353–364.
- Elster, J. (1998). Emotions and economic theory. *Journal of Economic Literature*, 36, 47–64.
- Elster, J. (1999). *Alchemist of the mind: Rationality and the emotions*. Cambridge, UK: Cambridge University Press.
- Fearon, J. & Laitin, D. D. (2003). Ethnicity, insurgency, and civil war. *American Political Science Review*, 97, 75–90.
- Fercello, C. & Umbreit, M. (1998). *Client evaluation of family group conferencing in 12 sites in 1st judicial district of Minnesota*. St Paul: Center for Restorative Justice and Mediation, University of Minnesota.
- Franks, D. D. & Gecas, V. (1992). Current issues in emotion studies. In D. D. Franks, & V. Gecas, (Eds.), *Social Perspectives on Emotion: A Research Annual*. Greenwich, CT: JAI Press.
- Frijda, N. (1994). "The Lex Talionis: On Vengeance,". In S. van Goozen, N. van de Poll, & J. A. Sergeant (Eds.), *Emotions: Essays on Emotion Theory*. Hillsdale, NJ: Lawrence Erlbaum.
- Frijda, N., Manstead, A. and Bem, S., eds. (2000). *Emotions and beliefs: How feelings influence thoughts*. Cambridge: Cambridge University Press.
- Gallagher, D., Clore, G. (1985) "Effects of Fear and Anger on Judgments of Risk and Evaluations of Blame," Paper presented at the Midwestern Psychological Association, May 1985.
- Gibson, J. L. (2004). *Overcoming apartheid: Can truth reconcile a divided nation?*. New York: Russell Sage Foundation.
- Goldberg, J. H., Lerner, J. S., & Tetlock, P. E. (1999). Rage and reason: The psychology of the intuitive prosecutor. *European Journal of Social Psychology*, 29, 781–795.
- Gonzalez, M. H., Haugen, J. A., & Manning, D. J. (1994). Victims as 'narrative critics.' Factors influencing rejoinders and evaluative responses to offenders' accounts. *Personality and Social Psychology Bulletin*, 20, 691–704.
- Gould, R. V. (2000). Revenge as sanction and solidarity display: An analysis of vendettas in nineteenth-century Corsica. *American Sociological Review*, 65(5), 682–704.
- Green, D. & Shapiro, I. (1994). *Pathologies of rational choice theory*. New Haven: CT: Yale University Press.
- Hamber, B. & Wilson, R. A. (2002). Symbolic closure through memory, reparation and revenge in post-conflict societies. *Journal of Human Rights*, 1, 35–53.

- Holtgraves, T. (1989). The form and function of remedial moves: Reported use, psychological reality, and perceived effectiveness. *Journal of Language and Social Psychology*, 8, 1–16.
- Hymans, J. E. C. (2006). *The psychology of nuclear proliferation: Identity, emotions and foreign policy*. Cambridge: Cambridge University Press.
- Kalyvas, S. (2006). *The logic of violence in civil war*. Cambridge: Cambridge University Press.
- Kassinove, H., Sukhodolsky, D. G., & Tsytarev, S. G. (1997). Self-reported anger episodes in Russia and America. *Journal of Social Behavior and Personality*, 12, 301–324.
- Kaufman, S. J. (2001). *Modern hatreds: The symbolic politics of ethnic war*. New York: Cornell University Press, Chapter 1– Chapter 2.
- Kaufmann, C. (1996). “Intervention in ethnic and ideological civil wars: Why one can be done and the other can’t,”. *Security Studies*, 6(1), 62–100.
- Keltner, D., Ellsworth, P., & Edwards, K. (1993). Beyond simple pessimism: Effects of sadness and anger on social perception. *Journal of Personality and Social Psychology*, 64, 740–752.
- Kirk, R. (2003). *More terrible than death: Massacres, drugs, and America’s War in Colombia*. New York: Public Affairs.
- Larsen, R. J. (1993) “Mood Regulation in Everyday Life,” In D.M. Tice (Chair), *Self-Regulation of Mood and Emotion*, Symposium conducted at the 101st Annual Convention of the American Psychological Association, Toronto, Canada.
- León, J. (2005). *País de Plomo: Crónicas de Guerra*. Bogotá: Aguilar.
- Lerner, J. S., Gonzalez, R. M., Small, D. A., & Fischhoff, B. (2003). Effects of fear and anger on perceived risks of terrorism: A national field experiment. *Psychological Science*, 14, 144–150.
- Lerner, J. S. & Keltner, D. (2000). Beyond valence: Toward a model of emotion-specific influences on judgment and choice. *Cognition and Emotion*, 14, 473–493.
- Lerner, J. S. & Keltner, D. (2001). Fear, anger, and risk. *Journal of Personality and Social Psychology*, 81, 146–159.
- Lerner, J. S. & Tiedens, L. Z. (2006). Portrait of the angry decision maker: How appraisal tendencies shape anger’s influence on cognition. *Journal of Behavioral Decision Making*, 19, 115–137.
- Loewenstein, G. (2000). Emotions in economic theory and economic behavior. *The American Economic Review*, 90, 426–432.
- Mano, H. (1994) “Risk-taking, Framing Effects, and Affect,” *Organizational Behavior and Human Decision Processes*.
- Mockus, A., former Mayor of Bogotá. (2007) “Why Forgiveness?” Seminario Internacional Paz y Responsabilidad en la Resolución de Conflictos, Bogotá June 16, 2007.
- Morris, W. & Rielly, N. (1987). Toward the self-regulation of mood: Theory and research. *Motivation and Emotion*, 11(3), 215–249.
- Newhagen, J. (1998). Anger, fear and disgust: Effects on approach-avoidance and memory. *Journal of Broadcasting and Electronic Media*, 42, 265–276.
- Ohbuchi, K., Kameda, M., & Agarie, N. (1989). Apology as aggression control: its role in mediating appraisal of and response to harm. *Journal of Personal Social Psychology*, 56, 219–227.
- Ortony, A., Clore, G. L., & Collins, A. (1988). *The cognitive structure of emotions*. Cambridge: Cambridge University Press.
- Petersen, R. (2001). *Resistance and rebellion: Lessons from Eastern Europe*. Cambridge: Cambridge University Press: Studies in Rationality and Social Change.
- Petersen, R. (2002). *Understanding ethnic violence: Fear, hatred, and resentment in twentieth century Eastern Europe*. Cambridge: Cambridge University Press.
- Petersen, R. (2005). Memory and cultural schema: Linking memory to political action. In F. Cappolletto (Ed.), *Memory and second world war: An ethnographic approach*. Oxford: Berg.
- Petersen, R. & Zukerman, S. (2009). “Revenge or Reconciliation: Theory and Method of Emotions in the Context of Colombia’s Peace Process,” forthcoming in the Peace Research Institute of Oslo’s Forum for International Criminal Justice and Conflict publication series. Spanish version to be published in Colombia by the Vice Presidency and the Universidad del Rosario Press.
- Petrucchi, C. J. (2002). Apology in the criminal justice setting: Evidence for including apology as an additional component in the legal system. *Behavioral Sciences and the Law*, 20, 337–362.
- Pinker, S. (2002). *The blank slate: The modern denial of human nature*. New York: Viking Press.
- Riker, W. (1986). *The art of political manipulation*. New Haven: Yale University Press.
- Rusting, C. L. & Larsen, R. J. (1993) “Negative Mood Regulation: Evidence for Specificity,” paper presented at the 101st Annual Convention of the American Psychological Association, Toronto, Canada.

- Rusting, C. & Nolen-Hoeksema, S. (1998). Regulating responses to anger: Effects of rumination and distraction on angry mood. *Journal of Personality and Social Psychology*, 74(3), 790–803.
- Scher, S. J. & Darley, J. M. (1997). How effective are the things people say to apologize? Effects of the realization of the apology speech act. *Journal of Psycholinguistic Research*, 26, 127–140.
- Schlenker, B. R. & Darby, B. W. (1981). The use of apologies in social predicaments. *Social Psychology Quarterly*, 44, 271–278.
- Sukhodolsky, D. G., Golub, A., & Cromwell, E. N. (2001). Development and validation of the anger rumination scale. *Personality and Individual Differences*, 31(5), 689–700.
- Suny, R. G. (2004) “Why We Hate You: The Passions of National Identity and Ethnic Violence.” February 1, 2004. *Berkeley Program in Soviet and Post-Soviet Studies*.
- Tajfel, H. & Turner, J. C. (1979). An integrative theory of intergroup conflict. In S. Worchel & W. G. Austin (Eds.), *The social psychology of intergroup relations*. Monterey, CA: Brooks-Cole.
- Tavuchis, N. (1991). *Mea culpa: A sociology of apology and reconciliation*. Stanford, CA: Stanford University Press.
- Thayer, R. E., Newman, J. R., & McClain, T. M. (1994). Self-regulation of mood: Strategies for changing a bad mood, raising energy, and reducing tension. *Journal of Personality and Social Psychology*, 67(5), 910–925.
- Tice, D. M. & Baumeister, R. E. (1993). Controlling anger: Self-induced emotion change. In D. M. Wegner & J. W. Pennebaker, eds., *Handbook of mental control* (393–409). Englewood Cliffs, NJ: Prentice Hall.
- Tripp, T. M. & Bies, R. J. (forthcoming). Righteous anger and revenge in the workplace: The fantasies, the feuds, the forgiveness. In M. Potegal, G. Stemmler, & C. Spielberger (Eds.), *A handbook of anger: Constituent and concomitant biological, psychological, and social processes*. New York: Springer.
- Tsebelis, G. (1990). *Nested games: Rational choice and comparative politics*. Berkeley, CA: University of California Press.
- Van Evera, S. (2001) “Primordialism Lives!” *Comparative Politics Section Newsletter*, Winter 2001.
- Villegas, G. Director of the Peace and Reconciliation Program, Office of the Mayor, Medellín. (2005). Untitled chapter. In C. J. Arnson (Ed.), *The peace process in Colombia with the Autodefensas Unidas de Colombia-AUC*. Washington, D.C: Woodrow Wilson International Center for Scholars.
- Weiner, B., Graham, S., Peter, O., & Zmuidinas, M. (1991). Public confession and forgiveness. *Journal of Personality*, 59, 281–312.
- Weinstein, J. (2007). *Inside rebellion: The politics of insurgent violence*. Cambridge: Cambridge University Press.
- Wood, E. (2003). *Insurgent collective action and civil war in El Salvador*. Cambridge: Cambridge University Press.

Subject Index

A

Acoustic

- analyses, 140–141, 143
- variables (attributes), 9, 58–59, 61, 64, 70, 146, 154, 218

Adrenocortical function, 181, 184–185, 442

See also Hormones, cortisol

Adrenomedullary system, 442

Affect, *see* Emotions

Aggression

- abuse cycle, 537–538
- affective, 45, 80, 82, 268, 274, 276, 278–279
- aggressive fantasies, 388–389, 396
- assault, 29, 31, 64, 81–82, 86, 89, 197–198, 211, 303, 332, 338, 351, 365, 392, 396, 468, 476–478, 481, 484, 500, 519, 536–538
- defensive (counteraggression), 45–46, 79, 231, 294, 428, 470, 542
- instrumental, 28, 45, 79–81, 231, 233, 237, 364, 406, 481
 - intervention and prevention programs for, 45, 485
 - in psychiatric patients, 53, 68, 73, 80–81, 86, 94, 407, 467, 469, 473, 478, 480, 486
- intimate partner, 4, 535
- offensive, 542
- physical, 4, 12–13, 16, 28, 45, 50, 64–65, 79, 81, 198, 224, 244, 351–352, 467, 485, 501, 518–520, 524, 526–528, 535
- post-ictal, 41, 50
- predatory, 44–45, 231
- proactive, 4, 28, 45, 80, 231–238
- reactive, 5, 45–46, 54–80, 196, 223, 231–238, 316, 443
- verbal, 13, 39–40, 46, 51, 64, 68, 79, 81, 84, 234, 237, 351–352, 355, 478, 519, 521

Agreeableness, 70, 89

Amok, 14, 466, 468

See also Berserker

Anger

- benefits of, 47, 52, 275, 279, 303, 368, 373, 376, 416, 453, 555, 575
- control or regulation of, 223–225

- costs of, 249, 277, 279, 303, 361–373, 423, 428, 468, 483, 576

- dyscontrol, 41–42, 465, 476–477, 486, 536

See also Anger intensity (degree, levels, magnitude)
of, out of control (OOC)

Anger as an approach emotion, 182

Anger and angry expressions

- attention to, 222, 317–318, 368, 470
- hostile attributional bias, 88, 223, 470
- perception of, 51, 132, 151, 256, 260, 314–318

Anger attacks, 478

Anger, components of

- action pattern, tendencies, 42, 177–181, 187, 541
 - anger-related behaviors, 64, 133–134, 198, 212, 312, 332
- bodily sensations, 104, 110–112, 282, 345
 - feeling hot, 104, 110
 - muscle tension, 105, 109
- coherence of components (measures), 172, 200–203
- facial expressions, 13, 17, 43, 53, 61–64, 126–127, 144, 149, 152, 154, 179, 183, 195–197, 210, 244, 249–253, 268, 280–281, 293, 297, 313, 315–319, 330, 387, 403
 - action units (AUs), 195, 250
 - cultural difference, 125, 131–133, 312, 388
 - mimicry, 51, 152–153
- psychophysiology, 3, 53–54, 104–105, 233
 - See also* Psychophysiology of anger, somatovisceral
- subjective experience (conscious feeling), 40, 54, 62, 150–151, 197, 209, 293, 350–352, 391, 394, 538
 - feeling angry, feeling annoyed, 5, 197–198, 330–333, 337, 340–341, 410
- vocal expression, 61, 139–154, 182, 195, 250–252, 332
 - fundamental frequency (pitch), 141–142, 153–154, 251
 - loudness, 149, 195, 197
 - prosody, 153, 251
 - rate, 142, 153
 - scream, 42–43, 200, 205, 207–209

- Anger, components of (*cont.*)
 shout, 41–43, 45–46, 50, 139, 194, 197, 200–201, 205, 207–209, 276, 391, 420, 521
 speech, 16, 113–114, 139–145, 149, 152–153
 yell, 139
- Anger, definitions of
 prototype model, 15, 165, 269
 schema, 168–169
 script, 17, 107, 330
- Anger, deflection of
 displacement, 278, 452, 575
 redirection, 45, 452, 473
- Anger, effects (on)
 attention, focus, 157, 330, 388
 cognition (thoughts), 127, 390
 concern van kleef, 545–556
 decision-making, 287–305, 386, 553
 distancing (psychological, social), 6, 132, 225, 466, 469
 fear suppression, 466, 485
 information processing, 6, 241–261, 449, 470, 480, 547, 552
 depth of processing (heuristic vs. systematic), 294, 298–299, 386
 interpretation of intent, 11–12, 14, 16, 21, 49, 51, 72–73, 95, 104, 108, 116, 140, 162, 208–209, 244, 249, 254, 270–271, 274, 278, 281, 313–320, 356, 385, 397, 417–418, 449, 501, 528, 564
 intrapersonal vs. interpersonal effects van kleef, 545–556
 judgment, 6, 11–12, 15, 129–130, 145, 147, 158, 165, 200, 272, 287–305, 310, 316, 417, 458, 500
 memory, 39, 50–52, 72–73, 91, 160, 245, 250, 255–256, 258, 275, 281, 289, 300, 303, 311, 390, 477, 566, 568, 571–573
 negotiation, 4, 225, 310, 545–556
 optimism, 4, 6, 18–19, 53, 65, 257–258, 294, 297, 301–304
 perception, 6–7, 39, 41, 48, 50–51, 53, 71–72, 103–104, 132–135, 144–145, 152–153, 165, 213, 219, 222, 250, 271, 281, 291, 296–297, 300, 314–320
 social, 87, 313, 315, 373
 perceived social status, 20, 39, 52, 90, 210, 225, 296, 329, 334, 342, 369, 371
 perspective taking, 208, 502
 prejudice, 296, 565–566, 571, 578
 punishment, 11–12, 15–16, 39, 47–48, 52–53, 225, 271–272, 291, 295–296, 303, 362, 394, 424, 471, 521–523, 529, 571, 573–578
 response (acts, behavior), 4, 466, 477, 484, 486
 risk-taking, 32, 297, 366, 368, 373
 sense of one's own increased energy, potency, empowerment, 19, 128, 303, 309, 344, 354, 358, 466–467, 478, 481, 505, 507, 531
 trust, 108, 129, 294, 296, 318, 335, 342, 479, 483, 536, 547
- Anger elicitation or induction, *see* Triggers
- Anger, episodes of, 194, 198, 210, 387–388
See also Tantrums
- Anger, escalation of, 387, 389–390
- Anger, functions of (goals of)
 adaptive (evolutionary), 15–16, 62, 151, 251–252, 312, 368, 390, 450, 485, 500
 communication (social signal), 48, 151, 210, 224, 249, 394
 of displeasure, personal state, social significance, 7, 149, 278, 405, 467
 goal blockage removal, 3, 181, 183, 220
 intimidation, 53, 390
 maintaining the social order, 128–129
 social dominance (control), 231
- Anger, genetics of
 heritability, 28–29, 374–375
 polymorphisms, 30, 374
 XYY syndrome, 30
- Anger-hostility-aggression (AHA) complex, 5, 40, 312–313
- Anger intensity (degree, levels, magnitude) of
 annoyance, 5, 62, 269, 330, 332, 405, 407, 539
 fury, 13–14, 269, 406, 468
 irritation, 62, 245, 269, 278, 406, 410, 539
 out of control (OOC), 53, 391
 rage, 5, 10–19, 116, 177–179, 195, 209, 269, 367, 386, 396, 404–406, 451
 blind, 5, 391, 471
 as different from anger, 5, 255
 as extreme form of anger, 19, 30, 194
 resentment, 10, 13, 81, 407, 425, 456, 570
 subanger, 5, 539–540
- Anger and the law
 insanity defense, 471–472
 mitigation, 471
 provocation defense, 386, 468, 471
 reasonable person (man) standard (test), 386, 471
 temporary insanity, 471–472
- Anger, priming of, 390
- Anger-prone, *see* Trait anger
- Anger, targets of
 deflection in pain patients, 453, 455–457, 459
 female vs. male, 4, 350, 366
 self vs. other, 104–105
- Anger, treatment (management) of
 cognitive behavioral affective therapy (CBAT), 505–506, 509
 cognitive behavioral therapy (CBT), 486, 504
 empty chair technique (Gestalt therapy), 502, 508
 intervention, 5, 17, 200, 316, 455
 outcome, 4, 30, 64, 149, 183–184, 207, 225, 254, 293, 301, 332, 353
 philosophico-religious approaches, 502

- prevention, 316, 500, 505–510
- rational-emotive therapy, 504
- relaxation, 279, 503–504, 508
- remediation (postvention), 500, 505–506
- response prevention, 507
- role play, 313, 426, 504, 507
- stress inoculation training, 504
- thought-stoppage, 389
- See also* Anger, control or regulation of
- Anger, triggers for (instigation of, provocations of, “causes”)
 - abandonment, 451, 467, 480
 - accidents, 273, 367, 369, 450
 - aggression, 53, 414
 - betrayal, 332
 - coercion, 350, 390, 486, 530
 - criticism, 42, 154, 335, 388, 416, 437, 521
 - deceit, 451, 481
 - experimental induction (elictation), 106, 108, 152, 258
 - exploitation, 53, 365, 451
 - frustration (goal blockage, *vs.* goal attainment), 43, 179, 200, 244, 258, 273, 275, 278, 343
 - See also* Frustrative non-reward
 - gossip, 349, 352, 366
 - imagery/imagination, 72, 107–108, 110–111, 258, 455
 - “real-life” manipulation, 105
 - incompetence of other(s), 332
 - injustice (unfairness, illegitimacy), 15, 18–19, 210, 248, 257–258, 272, 301, 343, 414–417, 422, 428
 - insult (status and power derogation), 13, 18, 40, 51, 69, 71, 178, 313, 358, 418
 - moral violations, 248, 254
 - neglect, 52, 151, 235, 255, 276, 354, 386, 419, 451, 469
 - norm violations, 19, 254, 259
 - rejection, 4, 12–13, 165, 221, 291, 316, 455, 469, 549
 - rushed for time, 335–337, 339
 - transgression, 4, 95, 361–376
 - nature of, 365
 - response to, 362–363, 366–368, 374–375
 - See also* Pain/physical discomfort; Stress
 - Berkowitz, Litvak; Threat
- Anger, typologies (kinds) of
 - cold *vs.* hot, 16, 42, 111, 147, 149, 172–173, 279, 481
 - cold anger, 142, 147, 149–150, 172–173, 269, 417, 421–422, 425, 428, 482
 - See also* Revenge (retaliation, vengeance)
 - constructive, malicious and fractious, 5
 - just (righteous), 10, 12–13, 18–19, 343, 414–415, 417, 421–422, 425, 428, 482
 - two types, 19, 255, 280, 296, 355, 363
- Anger, valence of, *see* Emotions, valence (positive *vs.* negative emotions)
- Anger-in *vs.* anger-out, 68, 92, 279, 351, 404–405, 460, 473
- Anticonvulsants, 91–96
- Antipsychotics, 92–93, 96
- Antisocial behavior
 - covert problem behaviors, 519
 - lying and stealing promise breaking, lying and surveillance avoidance, 522–523
 - externalizing (behavior, disorder, problems), 208, 221, 224–226
 - onset and growth of (developmental trajectories), 520
- Apology, 394, 397, 567, 573–574
- Appeasement, 11, 538
- Appraisal (models, construals, processes, causal analysis, judgment, assessment of motives & intents)
 - appraisals leading to anger *vs.* to other negative emotions, 6, 63, 74, 79, 220, 274, 291, 295, 304
 - appraisal-tendency framework (ATF), 288–291, 296, 298–301, 304
 - biases, *see* Biases
 - componential appraisal models (CPM), 245–247, 257, 260
 - of intent, 317, 319
 - malevolence, 417–418
 - selfishness, 417–418
 - naming, blaming, and claiming, 17, 417
 - social appraisal theory, 350, 352–354, 355–357
 - subconscious personification, 452, 458
- Approach-withdrawal, 74
- Aristotle, 15, 18, 21, 52, 302, 304
- Arm pull, 64, 186
- Arm restraint, of child, 179, 182, 197, 210, 390
- Arousal
 - regulation of, 223
- Articulated Thoughts in Simulated Situations (ATSS), 540–541
- Assertiveness *vs.* anger, 29, 65, 71, 89, 178, 182, 353–354, 504, 542
- Associative networks
 - affective associative networks, 300
 - neo-associationistic models, 6
- Ataque de nervios, 391
- Atherosclerosis
 - coronary artery calcification, 439, 441
- Atrocities
 - memories of, 389
 - survivors of, 568
- Attachment
 - fearful-angry, 536
 - insecure, 220, 224, 319, 469
- Attribution
 - fundamental error (dispositions *vs.* circumstances), 150, 288, 409, 547
 - of hostile intent, 316–317, 320
 - theory, 150, 271, 316, 507
 - See also* Anger, effects (on), judgment, perception

Autonomic nervous system (ANS), 27, 108, 112–113, 116, 161, 407
sympathetic division, 112, 115–116, 184–185

B

Beserker, *See also* Amok

Beta function, 202–204, 208

Biases

actor-observer, 418–420, 547
dispositions vs. circumstances, 317, 547

Blame (fault, responsibility)

of an external agent, 258, 271, 274–275, 470
nonemotional, 457
of others by pain patients, 453, 457, 459
of self, 357

Body size, 365, 371

See also Sex (gender), differences

Bosnia, 566, 568

Brain

amygdala, 40, 43–44, 46, 50, 53, 213, 368
brainstem, 44–45, 47, 112
cingulate, 32, 46, 49–50, 52, 72, 113, 116, 118
 anterior cingulate gyrus (ACG), 46–48, 53
cortex (cortical area), 4, 32, 46, 49, 65, 67, 72, 116, 118, 151, 212, 392
dorsal striatum, 303
dorsolateral frontal, 48, 72
executive control networks, 521
frontal, 4, 40, 46–47, 49, 52–54, 65–67, 69, 72–73, 302
hemispheric asymmetries (left vs. right), 40, 42, 212
hypothalamus, 44–45, 47, 52, 112
insula potegal, 49, 53, 113, 116, 118, 392
limbic emotion-motivation networks, 521
orbitofrontal (OFC), 46, 49, 212, 392
periaqueductal gray, 45, 213
prefrontal, 47, 49, 72–73, 116, 118, 303, 486
temporal, 39–40, 42, 46–47, 50, 52–54, 72, 115, 302
uncinate fasciculi, 47
ventromedial frontal (VMFC), 46–54

Bully, 225, 293

Buspirone, 84, 90–91

Bystander, 249, 428

C

Cardiovascular disease (CVD), 18, 149, 435–444, 460

Catatymic crisis, 395–396, 471–472

Catecholamines

dopamine (DA, dopaminergic), 85, 87
epinephrine (adrenalin), 443
 adrenaline-noradrenaline hypothesis, 116
norepinephrine (NE, noradrenaline), 32–33, 443
 alpha-adrenergic, 117
 alpha-methyl-para-tyrosine (AMPT), 85
 beta-adrenergic, 118
 MHPG, 84

Catechol-O-methyltransferase (COMT), 33–34

Catharsis, *see* Termination of anger

See also Anger, treatment (management) of; Venting

Children

abuse of (maltreatment), 537
child care, 3, 209, 212, 220, 353, 456
child rearing, 4
 aversive strategies nagging, criticism, nattering, 521
deviancy training, 522
discipline, 15–16, 20, 211, 319, 522–527, 529–530
 harsh, 522–526, 530, *See also* Punishment
parental investment, 366, 373
socialization, 42, 211, 220–221, 224–225, 319, 520
 social competence, children's, 220

Cholesterol

HDL, 439, 442

LDL, 438, 441

Cholinergic, 116–117

Clonidine, 84

Coercion, *see* Dominance, domination

Cognitive distortions, 482, 540, 563, 565

Cognitive-neoassociationistic model (CAN), 278

See also Appraisal (models, construals, processes, causal analysis, judgment, assessment of motives & intents)

Columbia

ELN guerrillas, 575

FARC, 566, 575

La Violencia, 5

Competition, 15, 52, 150–151, 165, 365–366, 368–369, 373, 376, 547, 554

Conflict

armed, 568
 cultural symbols in, 568
history of, biased punctuation, 418, 426–428
negative spiral (vicious cycle), 547

Confusion matrix, 147

Conscientiousness, 70, 83, 95

Context effects (experimental control), 49, 105–106, 110

Coping, 68–69, 149, 182, 201, 221, 224, 257, 259, 271–274, 276–277, 316, 395–396

 justification, 274, 423

Core affects, 7

Core relational themes, 45, 272

Coronary heart disease (CHD)

 psychosocial risk factors, 438
 cigarette smoking, physical activity, body mass index, and alcohol use, 438

Coronary heart disease (CHD), 297, 404, 436–441

Corrugator muscles, 126

Cross-cultural (cross-national) comparisons, 130–133, 141, 143–144, 154, 162–166, 354

 individualism vs. collectivism, 131–133

Crying, 196, 200, 211, 352–353, 356, 391, 395, 422

Cytokines, 443

D

- Darwin, 16, 64–65, 126, 178, 181–183, 186, 404, 485, 550
- Dementia, 7, 39–40, 42, 466, 484–485
- Deterrence, 363, 576
- Development, *see* Children
 - of emotional control, 480
- Display rules
 - maximization, 212
 - minimization, 212, 221
- Dominance, domination, 5, 21, 30, 44–46, 89, 185, 211, 278, 304, 315, 362, 366, 368, 564, 568

E

EEG

- alpha (EEG), 43, 68, 118
- frequency, 41, 43, 68
- power, 43, 68
- coherence, 71–72

Embodiment

- of anger, 158–159, 170–173
- of cognition, 153
- of emotion, 158–159

Emotions

- basic, 27, 112, 126, 143, 253, 312, 517
 - beliefs about, 223
 - comparison of, 110
 - contagion, of mood and emotions, 153, 281, 355, 550
 - cross-cultural comparison, 133, 140, 143, 154, 159, 162, 171, 195, 354
 - differential emotions theory (DET, discrete emotions), 7, 312, 520
 - diffuse (affect, mood), 5, 112, 178, 471, 546–547
 - dimensional models of, 245
 - emotional reactivity, 521, 526, 528–529
 - fear and anger, 65, 105, 111, 253, 290, 296–297, 319, 523
 - feelings, 5, 45, 68, 111, 152, 196, 222, 244, 256, 269, 330, 342, 405, 458, 503
 - intensity of, 249, 332, 572–573, 576–577
 - interactions of, 51, 523, 528–529
 - prototype theory, 7, 15, 42, 165, 269, 276
 - schema, 111, 312, 314, 316
 - as a switch, 92, 392, 542, 570–571
 - valence (positive vs. negative emotions)
 - of anger, 67, 313
 - vocal expression of, 140, 142, 149–152
- Emotions, moods and states (other than anger), specific
- anger-like emotions, 330
 - anxiety, 72, 88, 94, 106, 147, 280, 316, 408, 466, 476, 503
 - disappointment, 43, 350
 - disgust, 48–49, 108, 144, 148, 567
 - distress, 7, 95, 179, 202, 393, 450, 479
 - elation, 142, 147–148
 - euphoria, 65

- fear, 7, 45, 65, 95, 107, 110, 196, 261, 280, 319, 450, 500
 - grief, 14, 162, 456, 567, 573
 - bereavement, 473
 - guilt, 14, 254, 259, 294, 567
 - happiness, 48–49, 63, 65, 147, 224, 298, 303, 330, 552
 - irritability, 89, 92, 479
 - jealousy, 469, 538–539
 - joy, 64, 104, 127, 149, 162, 256
 - pride, 6, 255, 294, 456, 565
 - remorse, sorrow, 42, 394, 481, 574
 - sadness, 7, 43, 107, 147, 152, 180, 184, 197, 280, 294
 - shame, 148
- Empathy, 47, 52, 177–178, 482
- Epilepsy
 - hypermotor, 46
 - temporal lobe epilepsy (TLE), 41, 396
- Epistemic motivation, 552, 554
- Ethnic identity, 561
- Excitation transfer, 387
- Executive processes (control), 15, 116, 518, 520–524, 526–527, 529–530
- Extinction, 64, 66, 180, 274, 503

F

- Family, 11, 13, 20, 32, 47–48, 86, 130–131, 209, 221, 317, 339, 342, 353, 421, 456, 476, 520–526, 529–530, 539–540
- Feedback (self-produced cues)
 - facial, 6, 40, 53, 280, 290, 390, 458
 - muscular movements, 282
 - vocal, 390, 503
- Feedforward, 40
- Fenfluramine, 32, 82–84, 93, 95, 392
- Fight vs. flight, 45–46, 48, 66, 115, 118, 126, 235, 244, 249, 277–278, 291, 303, 564
 - See also* Aggression
- Forgiveness, 394, 413–428, 576, 578
- Freud, 15, 177, 195, 303, 394–395, 404, 472–473, 476, 502
- Frustrative non-reward, 521, 523
- Functionalist Approach, 151, 531
 - See also* Anger, functions of (goals of)

G

- GABA (gamma aminobutyric acid), 50
- Galen, 16, 465
- Game theory, 273, 292
- Goal blockage, *see* Anger, triggers for (instigation of, provocations of, “causes”), frustration (goal blockage, vs. goal attainment)
- Growth models, 525

H

- Headache, 167, 275, 279, 460, 473
- Heart rate, heart rate variability
 - See also* Autonomic nervous system (ANS)

Homicide (murder)
 spree murder, 468
See also Aggression

Hormones

adrenocorticotropin, 443
 cortisol, 82, 84, 297, 443
 estrogen, 29
 growth hormone (GH), 82, 84, 370
 prolactin (PRL), 82, 84
 testosterone, 28

Hostility, 5, 28, 31, 48, 63, 80–83, 85–90, 92–95,
 184, 244, 256, 282, 390, 404–407, 409–410,
 435–444, 460, 472–477

Hypertension, 184, 404, 409–410, 442, 473

Hypothalamic-pituitary adrenal (HPA) axis, 442

I

Impulsivity, 32–33, 68, 79, 82–83, 276, 367–368,
 370–371, 481–482, 486, 521, 537

Individual differences, *see* Personality, traits

Insanity, 14, 16, 466, 468, 471–472

See also Psychopathology

J

Justice

interactional, 416
 organizational, 414, 416
 procedural, 425–426
See also Anger, triggers for (instigation of,
 provocations of, “causes”), injustice
 (unfairness, illegitimacy)

L

Language

development of, 224
 linguistic metaphors for anger, 4, 160, 164
 pressurized container, 158–163, 168, 170, 172–173
 angry breath, 104, 110, 508
 heat, hot fluid, 158, 160–163, 165, 172
 metonymies, 161–162, 168, 172
 words for anger in other languages, 171

Learned helplessness, 64, 277

Libido (life instinct), 177, 404

Lifespan, anger over, 370

age effects, 169

List from T&B

stereotyping, 299

See also Appraisal (models, construals, processes,
 causal analysis, judgment, assessment of
 motives & intents)

M

MA50, 194, 207–209

Meta-chlorophenylpiperazine (m-cpp), 83

Minnesota Twin Study, 33

Momentary Anger [MA(t)], 194, 202–206, 208–213

Monoamine oxidase-A (MAO-A), 32–34

Motor theory of perception, *see* Embodiment

Multidimensional scaling (MDS), 144–145, 147–148

Myocardial infarction (MI, heart attack), 436, 500

N

Negative emotionality, 27, 185, 221, 224, 482, 526,
 536, 541

Negotiation

conciliation, 551, 553, 555
 demands, 128
 distributive, 551
 exchange relationships, 555
 mismatching, 551
 positions, 28, 152, 501
 strong, 80, 245, 333
 weak, 551, 553
 ultimatum offers, 549

Neoassociationist theories, *see* Associative networks

Nickel-and-dimed, 363

P

Pain/physical discomfort, 265, 275, 279, 450–453, 457
 aversive events/states, 150, 269, 273, 521

chronicity of, 461

cold pressor, 279, 459–460

Peer relations

acceptance, 221
 rejection, 4, 220, 225

Perpetrator (harmdoer, offender, transgressor,
 wrongdoer), 303, 362–363, 367, 395, 421,
 424, 451–453

Personality

externalizing, 7, 387, 466, 469, 480
 internalizing, 387
 need for cognitive closure, 552
 negative emotionality, 27, 482, 536, 541
 pain-prone personality, 460
 traits, 28–30, 33–34, 88, 258, 375, 425–426, 481–482
 type A, 18, 256, 336
 type B, 33, 404

Plato, 15, 18, 20

Power, *see* Dominance, domination

Pre vs. post-goal positive affect, 63

anticipatory pleasure of anger, 303

Processing, automatic vs. controlled, 392

Propranolol, 91, 94–95, 117

Psychopathology

anxiety disorders, 468, 473, 475, 479
 generalised anxiety disorder (GAD), 88, 90,
 95, 475
 panic attacks, 93, 391, 473, 477
 bipolar disorder, 42, 194, 467, 475
 culture bound syndromes, 468
 exotic psychosis, 468
 See also Amok; Berserker
 depression, 7, 41, 183, 466, 473, 475, 482

- intermittent explosive disorder. (intermittent episodic dyscontrol), 7, 41, 466
 - mania (hypomania), 65, 70, 474
 - personality disorders (Axis II), 479–481, 541
 - borderline personality disorder, 83, 92, 386, 396, 480, 537
 - borderline personality organization (BPO), 480, 537–538
 - narcissism, 177–179
 - post traumatic stress disorder (PTSD), 7, 84, 88, 91, 94–95, 278, 368, 453, 475–476
 - premenstrual dysphoric disorder (PMDD), 88, 91–92
 - psychoses (psychotic disorder), 469
 - command hallucinations, 470
 - delusional disorder (persecution), 469
 - paranoia, 91, 469, 479
 - schizoaffective disorder, 31, 469
 - schizophrenia, 467, 469
 - Psychophysiology of anger
 - blood pressure, 4, 104–107, 116, 233
 - diastolic, 105–110, 115–117, 184, 210, 437, 442, 469
 - systolic, 105–106, 109, 118, 184, 460, 469
 - cardiac, 105–109, 116, 184, 437–438, 443
 - output, 105–109, 116, 180
 - variability, 180, 185–186
 - facial flushing (redness), 40, 46, 197, 199
 - respiration, 105, 109–110, 116, 133, 161, 450
 - skin conductance, 105, 108
 - skin temperature, 105, 107–108, 116
 - somatovisceral, 103–105, 108, 116
- Punishment, 11–12, 15–16, 47–48, 52–53, 225, 271–272, 295–296, 303, 362, 424, 471, 521–523, 529, 531, 567, 573–578
- R**
- Race (race differences), 74, 335, 437, 439
 - Rational choice (RC) models, 563
 - individual strategies, 563
 - Reactance, 64, 467
 - Rebels, 564
 - Reconciliation, 355, 357, 415, 418, 423–425, 555, 561, 567–569, 573–578
 - Reinforcement, 65–66, 220, 421, 455, 504, 521, 523
 - Reparation, 13, 115, 500, 504, 575–578
 - Repression
 - political, 562, 575
 - psychological, 562
 - Reproductive fitness, 365–366, 372–374
 - Reputation, 3, 18, 45, 53, 363, 366, 370–371, 376, 416, 424, 553
 - Revenge (retaliation, vengeance)
 - avenger, 418–419, 423–424, 428
 - fantasies, 423–424
 - See also* Time, rumination
 - plotting, 424
 - revenge as justice, 414
 - Reward
 - appetitive, 63
 - consummatory, 63
- S**
- Scales, self-report (instruments, measures, questionnaires)
 - affect adjective checklist (AAC), 85, 540
 - Buss–Durkee hostility inventory, 27, 29, 31, 80–82, 89
 - Buss–Perry, 27, 29, 31, 80–82, 89
 - conflict tactics Scale, 537, 540
 - dyadic adjustment scale, 542
 - minnesota multiphasic personality inventory (MMPI)–2, 409, 440
 - anger content scale, 440
 - multidimensional anger inventory (MAI), 538
 - multiple affect adjective checklist (MAACL), 85–86
 - PANAS, 89
 - profile of mood states (POMS), 86–88
 - social desirability, 63, 538
 - state-trait anger scale (STAS), 83, 85, 405, 407–411
 - S-anger, 408
 - state-trait anger expression inventory (STAXI), 197, 405
 - T-anger, 408–409
 - Seizures, *see* Epilepsy
 - Seneca, 11, 15–16, 20, 404, 469, 472
 - Serotonin (5HT)
 - 5-HIAA, 80–81
 - acute phenylalanine/tyrosine depletion (APTD), 85, 87
 - fluoxetine, 91, 94
 - para-chlorophenylalanine (PCPA), 85
 - SSRI (citalopram), 89
 - transporter (5-HTTLPR), 31
 - tryptophan, 30, 85–89, 95
 - tryptophan hydroxylase (TPH), 30, 85
 - Sex (gender)
 - differences, 16, 28, 211–212, 350–357, 363–367
 - stereotypes, 350–353
 - Slow burn, 159, 386–387, 389
 - Social information processing (SIP) models, 47, 51, 222, 311–320, 397, 470, 480
 - Socialization, *see* Children, child rearing
 - Social Role Theory, 350, 352–353
 - traditional vs. egalitarian, 19, 210, 254, 354, 357, 436
 - Socioeconomic status
 - age, 196, 332, 396, 441
 - See also* Lifespan, anger over education schieman, 334, 339
 - income, 339, 342
 - feeling underpaid, 342–343
 - financial resources, 342
 - poverty, 54
 - See also* Stress Berkowitz, Litvak
 - Sociological analysis, 329

- Spinal motorneurons, 213
 size principal, 213
- State anger
 irate, 13, 542
 mad, 11, 14, 16, 196, 223, 236, 333, 422
 rabid, 10, 14, 468
 synonyms for, 501
 See also Language, Linguistic metaphors for anger
- Strength, *see* Dominance, domination
- Stress Berkowitz, Litvak, 4–6, 15, 287–305, 391
 social and economic, 279
- Stroke, 39, 42, 105–109, 213, 294, 436, 439, 441
- Sympathy, 69, 394
- T**
- Tantrums, 6, 16, 42–43, 46, 193–213, 224, 312, 387, 391–395
- Termination of anger
 catharsis, 393–395
 decay, 393, 567
 quenching, 393–394, 567
- Thanatos (death instinct), 404
- Threat, 5, 12, 45, 90, 95, 126, 149, 151, 181, 213, 222, 244, 253, 277–279, 312, 314–320, 362, 390, 469
- Time
 course (persistence) of anger, 198, 204, 209, 386
 desistance, 531
 duration, 204, 209
 pressure, 4, 508
 rise and fall, 5, 54, 194
 rumination
 discounting, 16, 362–363, 367
 forward vs. backward reflection, 302–303
 perspectives, (past vs. future), 388–389
- Tit for tat, 5, 428, 561
- Total peripheral resistance (vascular resistance), 4, 105–109, 116
- Trait anger
 high (anger-prone), 7, 54, 70, 256, 260, 304, 314–315, 436, 438–439
 low, 257, 304, 313–314
- Trauma, 91, 94–95, 371, 395, 466, 471, 476–477, 486, 537–538, 578
- Traumatic brain injury (TBI), 39, 41
- Triggers, 65–66
- Triglycerides, 442
- Trust, 129–130, 294, 296, 335–336, 342, 422, 547
- Tyrosine hydroxylase, 85
- V**
- Vagal
 brake, 186
 tone, 116
 withdrawal, 109, 117–118, 185–186
- Validity
 convergent, 108, 235
 discriminant, 108, 235
- Venting, *see* Catharsis
- Verbal ability, 222, 526
- Victims, 211, 225, 367, 369, 416, 418, 420–426, 453, 468, 476–477, 566
- Vigilantism, 426
- Vignettes (scenarios), 143
- Violence
 escalation of, 387, 542
 spiral model, 569
 experience of, 52, 158–159, 173, 303, 370, 374
 intensity of, 41, 54, 249, 364, 567
 interpersonal, 313, 316, 390, 474, 537
 “greed” theory, 564
 political, 562–574, 578, *See also* Wars
 post-violence period, 568
 See also Aggression
- Visceral factors, 16, 392
- W**
- Wars
 civil, 19, 562–565, 574
 interstate, 562
- Workplace, anger in, 343, 389